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RESPIRATORY CARE

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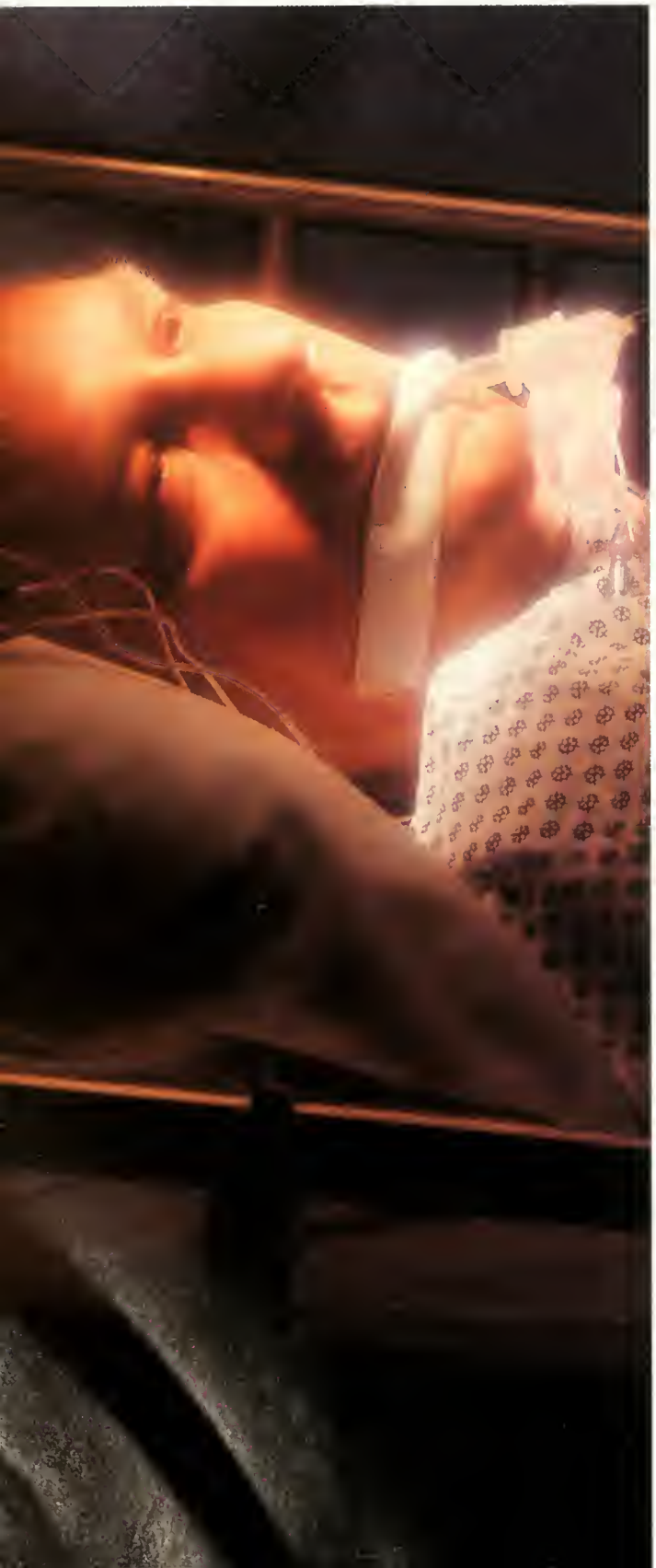

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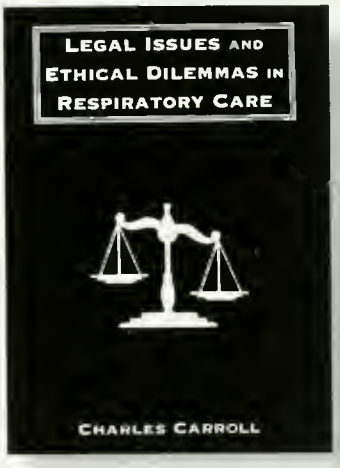

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CORRECTION

201 Figure 4 omitted
Del Bufalo C, Morelli A, Bassein L, Fasano L, Quarta CC, Pacilli AMG, Gunella G. Severity scores in respiratory intensive care: APACHE II predicted mortality better than SAPS II. Respir Care 1995;40(10):1042-1047.

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Editorials, Commentaries, and Reviews To Note

How To Ensure the Continued Resurgence of Tuberculosis (viewpoint)—LB Reichman. Lancet 1996;347:175-177.

Patients' Preferences and Randomised Trials (Essay)—WA Silverman, DG Altman. Lancet 1996;347: 171-174.

Cardiac Pacing (Review)—FM Kusumoto, N Goldschlager. N Engl J Med 1996;334(2):89-98.

Obstructive Sleep Apnea (Current Concepts)—PJ Strollo Jr, RM Rogers. N Engl J Med 1996;334(2): 99-104

Single-Breath Carbon Monoxide Diffusing Capacity (Transfer Factor) Recommendations for a Standard Technique—1995 Update (Statement)—The American Thoracic Society. Am J Respir Crit Care Med 1995;152:2185-2198.

Survivors of Extracorporeal Membrane Oxygenation at 1 Year of Age: The Relationship of Primary Diagnosis with Health and Neurodevelopmental Sequelae—J Bernbaum, IP Schwartz, M Gerdes, JA D'Agostino, CE Coburn, RA Polin. Pediatrics 1995;96(5):907.

OBJECTIVE: Although extracorporeal membrane oxygenation (ECMO) has been responsible for the improved survival of infants with cardiorespiratory failure, its use over the last decade has raised concern as to the health of the survivors and the severity of neurodevelopmental sequelae. Though infants meeting ECMO criteria have a variety of reasons prompting the use of this therapy, most studies to date have simply reported outcome on the entire population that has survived without regard to the original nature of the child's illness. The purpose of this study was to determine the type and extent of health-related problems and neurodevelopmental sequelae in infants requiring ECMO therapy and the association of these findings with the infants' primary diagnosis. METHODS: Eighty-two neonates required ECMO therapy between May 1990 and December 1993. The most common diagnoses prompting ECMO therapy included 26% with meconium aspiration syndrome, 34% with congenital diaphragmatic hernia (CDH), 16% with persistence of the fetal circulation, and 9% with sepsis. Information concerning the hospital course was obtained through chart review, and the infants were seen at 6 and 12 months of age for medical and neurodevelopmental follow-up. Data were analyzed using descriptive statistics and Fisher's exact test, *t*-tests, and analysis of variance where appropriate. Assessment of hospital course and discharge data focused on the 4 main diagnostic groups, whereas follow-up data were further limited to the 2 most frequently encountered groups (meconium aspiration syndrome and CDH). RESULTS: Overall survival was 79%. Significant differences in survival were noted based on primary diagnostic category. Those with CDH fared the worst, with an overall survival rate of 68% and a more complicated hospital course with a longer duration of ECMO. At discharge, the CDH group demonstrated a greater incidence of bronchopulmonary dysplasia, gastroesophageal reflux, feeding dysfunction, and hypotonia. No significant differences were noted in the incidence of intraventricular hemorrhage, cerebral infarction, extra-axial fluid collection, or seizures. Hearing loss was uncommon. During the first year of life, although no differences were noted in growth rate, infants in the CDH group continued to experience a higher incidence of gastroesophageal reflux (43%) and feeding dysfunction, with 36% of this group requiring tube feedings for nourishment. Although 40% of the entire ECMO population was diagnosed with bronchopulmonary dysplasia before initial discharge, by 1 year of age, 50% of those with CDH versus 17% of those with meconium aspiration syndrome continued to be clinically symptomatic. Although the ECMO population as a whole scored in the normal range developmentally, CDH infants had significantly lower motor and slightly lower cognitive scores at 1 year of age. Despite finding abnormal muscle tone in a high percentage of the entire ECMO population at discharge, most demonstrated resolution by 1 year of age. Of the CDH infants, however, 75% continued to evidence some degree of hypotonicity, which affected acquisition and quality of gross motor skills. CONCLUSION: Despite the impact that ECMO has had on the survival of infants with severe respiratory failure, the efficacy of ECMO cannot be assessed accurately without an analysis of the extent and morbidity in the surviving population. Most centers are reporting relatively low morbidity for the entire ECMO population. However, upon separating this population into primary diagnostic categories, we found that the CDH population encountered a greater number of neurodevelopmental, respiratory, and feeding abnormalities during the first year of life. The reasons for these differences are unclear but may be related to the severity of the primary illness itself or the variables associated with prolonged ECMO therapy. Stratifying outcome by primary diagnosis gives the health care provider more information to improve existing intervention techniques and to provide parents with more accurate counseling.

A New Device for Pleural Drainage in Newborn Infants—B Wood, M Dubik. Pediatrics 1995;96 (5):955.

OBJECTIVE: We tested a chest tube device and insertion technique designed to reduce complications and expedite effective thoracostomy tube placement. METHODOLOGY: This is a case series study. We placed chest tubes using this device in 25 infants ranging in weight from 425 to 3,500 g. This investigation was performed in a regional referral center neonatal intensive care unit. RESULTS: Using this device, chest tube insertion was safe and effective. No complications were identified. CONCLUSION: We believe percutaneous placement of small-bore pigtail catheters using this technique represents a useful alternative to the standard approach of thoracostomy tube placement in small infants. SYNOPSIS: To reduce

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complications during thoracostomy tube placement and to expedite tube insertion, we modified a chest tube device for use in premature infants. This technique and device facilitates quick and accurate placement. We placed chest tubes using this device in 25 infants ranging from 425 to 3,500 g. Using this device, chest tube insertion is quick, easy, and safe. No complications were identified. We believe percutaneous placement of small-bore pigtail catheters using this technique represents a useful alternative to the standard approaches of thoracostomy tube placement in small infants.

The Response of Flow-Triggered Infant Ventilators—M Nishimura, D Hess, RM Kacmarek. Am J Respir Crit Care Med 1995;152:1901.

Patient-triggered ventilation (PTV) has not been feasible for infants because of large trigger pressures and long delay times with pressure-triggered systems. Recently, 4 infant ventilators with flow triggering have become available. We questioned if delay times, trigger pressures, and trigger work with these ventilators would be acceptable for PTV in infants. All ventilators were attached via 3-, 4- and 5-mm endotracheal tubes to a spontaneously breathing infant lung model. The lung simulator was set at an inspiratory time of 0.65 s, tidal volume of 15, 30, and 45 mL, and 0 and 5 cm H_2O positive end-expiratory pressure (PEEP). Delay time, trigger pressure, and trigger work were determined from pressure measured at the proximal airway, trachea, and alveolus. There were significant differences between the endotracheal tube sizes, sites of measurement, ventilatory demand and ventilator brand at each PEEP level for delay time, trigger pressure, and trigger work ($p < 0.001$). Delay time was greatest with the 3-mm endotracheal tube at high ventilatory drive (maximum 138.2 ± 2.1 ms). Both trigger pressure (minimum 0.23 ± 0.02 cm H_2O) and trigger work (minimum 0.05 ± 0.01 g · mL) increased with decreasing endotracheal tube size, increasing ventilatory demand, use of PEEP, and site of measurement: alveolus > trachea > airway (maximum: trigger pressure 5.04 ± 0.02 cm H_2O; trigger work 114.48 ± 0.88 g · mL). PTV may not be appropriate under conditions of increased ventilatory drive and small endotracheal tube size in infants.

Score for Neonatal Acute Physiology: Validation in Three Kaiser Permanente Neonatal Intensive Care Units—GJ Escobar, A Fischer, DK Li, R Kremers, MA Armstrong. Pediatrics 1995;96(5):918.

BACKGROUND: Measurement of the severity of illness is a research area of growing importance in neonatal intensive care. Most severity of illness scales have been developed in tertiary care settings. Their applicability in community neonatal intensive care units has not been tested. OBJECTIVES: Our goal was to assess the operational characteristics of the score for neonatal acute physiology (SNAP): the relationship to birth weight, the length of total hospital stay, and in-hospital mortality. METHODS: We assigned SNAP scores prospectively to all inborn admissions at 3 community neonatal intensive care units during an 11-month period. Data on other neonatal predictors (eg, birth weight and the presence of congenital heart disease) were also collected. We measured in-hospital mortality, the experience of interhospital transport to a higher level of care, and total hospital stay. RESULTS: We found that the SNAP's relationship to birth weight was similar to previous reports. The SNAP's perinatal extension is a reliable predictor of newborn in-hospital mortality, with an area under the receiver operator characteristic curve of 0.95. The SNAP is also a good predictor of total hospital length of stay, whether by itself (by which it can explain 31% of the total stay) or in combination with other variables. Its predictive ability is better among infants of low birth weight (< 2,500 g) than among those of normal birth weight (≥ 2,500 g). The SNAP's predictive power was most limited among infants admitted to rule out sepsis. The predictive ability of a model containing birth weight, the SNAP, and transport status was not improved by the inclusion of 2 major diagnostic categories, the presence of congenital heart disease or complex illness. CONCLUSION: Although it has definite limitations among infants who weigh 2,500 g or more, the SNAP is a potent tool for outcomes research. Modification of some of its parameters could result in a multifunctional scale suitable for use with all birth weights.

Tobacco Information in Two Grade School Newsweeklies: A Content Analysis—ED Balbach, SA Glantz. Am J Public Health 1995;85 (12):1650.

OBJECTIVES: This study compared tobacco-related articles from 2 elementary school publications, *Weekly Reader* and *Scholastic News*, published in 1989 through 1994. METHODS: Articles for grades 4 through 6 were evaluated, and the publications were compared with each other. Also, issues of *Weekly Reader* published after acquisition by K-III, which is owned by the firm that formerly owned RJR Tobacco, were compared with the earlier ones. RESULTS: *Weekly Reader* was less likely than *Scholastic News* to mention short-term consequences of smoking (32% vs 64%) or to give a clear "no-use" message (35% vs 79%). *Weekly Reader* was more likely to give the tobacco industry position (68% vs 32%). Post-K-III issues of *Weekly Reader* were less likely to provide a clear no-use message than earlier ones (62% vs 24%). CONCLUSIONS: Health professionals need to monitor the health information carried in these publications, which reach between 1 and 2 million students per grade level each week. Although neither publication had perfect tobacco coverage, *Scholastic News* was significantly better than *Weekly Reader*.

Effectiveness of Bystander Cardiopulmonary Resuscitation and Survival Following Out-of-Hospital Cardiac Arrest—EJ Gallagher, G Lombardi, P Gennis. JAMA 1995;274:1922.

OBJECTIVE: To examine the independent relationship between effectiveness of bystander cardiopulmonary resuscitation (CPR) and survival following out-of-hospital cardiac arrest. DESIGN: Prospective observational cohort. SETTING: New York City. PARTICIPANTS: A total of 2,071 consecutive out-of-hospital cardiac arrests meeting Utstein criteria. INTERVENTION: Trained prehospital personnel assessed the quality of bystander CPR on arrival at the scene. Satisfactory execution of CPR required performance of both adequate compressions and ventilations in conformity with current American Heart Association guidelines. MAIN OUTCOME MEASURE: Adjusted association between CPR effectiveness and survival. Survival was defined as discharge from hospital to home. RESULTS: Outcome was determined on all members of the inception cohort—none were lost to follow-up. When the association between bystander CPR and survival was adjusted for effectiveness of CPR in the parent data set (n = 2,071), only effective CPR was retained in the logistic model (adjusted odds ratio [OR] = 5.7; 95% confidence interval [CI], 2.7 to 12.2; $p < 0.001$). Of the subset of 662 individuals (32%) who received bystander CPR, 305 (46%) had it performed effectively. Of these, 4.6% (14/305) survived vs 1.4% (5/357) of those with ineffective CPR (OR = 3.4; 95% CI, 1.1 to 12.1; $p < 0.02$). After adjustment for witness status, initial rhythm, interval from collapse to CPR, and interval from collapse to advanced life support, effective CPR remained independently associated with improved survival (adjusted OR = 3.9; 95% CI, 1.1 to 14.0; $p < 0.04$). CONCLUSION: The association between bystander CPR and survival in out-of-hospital cardiac arrest appears to be confounded by CPR quality. Effective CPR is independently associated with a quantitatively and statistically significant improvement in survival.

Hospital and 1-Year Survival of Patients Admitted to Intensive Care Units with Acute Exacerbation of Chronic Obstructive Pulmonary Disease—MG Seneff, DP Wagner, RP Wagner, JE Zimmerman, WA Knaus. JAMA 1995;274 (23):1852.

OBJECTIVE: To describe outcomes and identify variables associated with hospital and 1-year survival for patients admitted to an intensive care unit (ICU) with an acute exacerbation of chronic obstructive pulmonary disease (COPD). DESIGN: Prospective, multicenter, inception cohort study. SETTING: Forty-two ICUs at 40 US hospitals. PATIENTS: A total of 362 admissions for COPD exacerbation selected from the Acute Physiology and Chronic Health Evaluation (APACHE) III database of 17,440 ICU admissions. MEA-

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Saturday, December 2, 1995

☐	AARC95-001a	Keynote Address: Wake Up America! *William C. Dement MD, PhD*
☐	AARC95-003	Lung Volume Reduction Surgery *Joshua O Benditt, MD*
☐	AARC95-004	Cardiology for Automated External Defibrillation Providers *William Kaye, MD*
☐	AARC95-005	Advances in the Use of High Frequency Jet Ventilation *Sherry E. Courtney, MD, MS*
☐	AARC95-006	New Modes and Expanded Capabilities of Perinatal-Pediatric Mechanical Ventilators *Mark J. Heulitt, MD*
☐	AARC95-007	Advances in the Use of High Frequency Oscillatory Ventilation *Sherry E. Courtney, MD, MS*
☐	AARC95-008	Surviving a Joint Venture by Identifying the Crucial Role of the RCP on Transport Teams *Jerry A. Focht, RRT*
☐	AARC95-009	Inter/Intra Hospital Transport *Jamie L. Taylor-Irizarry, RRT*
☐	AARC95-010	Flight Physiology *Mark J. Heulitt, MD*
☐	AARC95-011	Air Transport of Trauma Victims *Jerry A. Focht, RRT*
☐	AARC95-012	Natural History of COPD *Barry Make, MD*
☐	AARC95-013	Scientific Basis for Current Therapy *Stephen I Rennard, MD*
☐	AARC95-014	Survival Following Severe Exacerbations of COPD *Joshua O Benditt, MD*
☐	AARC95-015	Outcomes After Exacerbations of COPD (Support Study) *Alfred F Connors Jr , MD*
☐	AARC95-016	New Approaches to Treatment *Stephen I Rennard, MD*
☐	AARC95-017	Transformational Leadership *Sam P Giordano, MBA, RRT*
☐	AARC95-018	Overcoming the Barriers *John Wright, MBA, RRT*
☐	AARC95-019	Care Plan Development *John S Sabo, MS, RRT*
☐	AARC95-020	Compiling Objective Data *John Wright, MBA, RRT*
☐	AARC95-021	What Is ARDS, Who Gets It, and What Happens to Them? Has Outcome Changed? *Kenneth P Steinberg, MD*
☐	AARC95-022	What Is Appropriate Ventilator Management Today? *Neil R MAcIntyre, MD*
☐	AARC95-023	Do Corticosteroids (or Other Drugs) Alter the Course of ARDS? *Kenneth P Steinberg, MD*
☐	AARC95-024	What Happens to ARDS Survivors? *Neil R MacIntyre, MD*

Sunday, December 3, 1995

☐	AARC95-027	What Have We Learned about Lung Injury from the Management of Near-Drowning? *Jerome H Modell, MD*
☐	AARC95-028/29	RCPs Meet the Challenge of the NAEPP, Urban Message--Getting the Message Out and Breaking the Barriers to Care *Thomas J Kallstrom, RRT; Alvin V Thomas, MD*
☐	AARC95-030	School-Based Asthma Management--Strategies for Asthma Care for the Child in School & Panel Discussion *Mark W Millard, MD*
☐	AARC95-031	Management of Acute Respiratory Failure without Intubation *Robert M Kacmarek, PhD, RRT; David J Pierson, MD*
☐	AARC95-032	Changing the Paradigm: The Role of the RCP in Health Promotion/Disease Prevention *William F Galvin, MSEd, RRT, CPFT*
☐	AARC95-033/34	RCPs in the Operating Room; RCPs on the Resuscitation Team *Richard D Branson, RRT; Charles G Durbin, Jr., MD*
☐	AARC95-035/36	RCPs in the Hyperbaric Chamber; RCPs in the Subacute Care and Skilled Nursing Facility Setting *John M Grayheal, CRTT; Dianne L. Lewis, MS, RRT*
☐	AARC95-037	Laboratory and Clinical Experience in Liquid Ventilation *Ronald B Hirschl, MD*
☐	AARC95-038	Practical Considerations in the Therapeutic Administration of Inhaled Nitric Oxide *Robert M. Kacmarek, PhD, RRT*
☐	AARC95-039	Tracheal Gas Insufflation *Sue A Ravenscraft, MD*
☐	AARC95-040	Ventilator Management to Avoid Barotrauma *David J Pierson, MD*
☐	AARC95-041	Gene Therapy for Cystic Fibrosis *David E. Geller, MD*
☐	AARC95-044	What Are Outcomes and Why Are They Needed? *Jeffrey A Souza, RN*
☐	AARC95-045	What Outcomes To Measure and How *Vianna Zimbel, BS, CRTT*
☐	AARC95-046a/b	Outcomes Measurement--What Lies Ahead *Jeffrey A Souza, RN* **and** Panel Discussion--Outcomes Measurement in Home Care
☐	AARC95-047	Guidelines for Predicting Mortality and Morbidity in Cardiac Disease *Michael B Higginbotham, MD*
☐	AARC95-048	Preoperative Assessment in Upper Abdominal and Thoracic Surgery *Charles G Irvin, PhD*
☐	AARC95-049	Maximizing Exercise Performance in Geriatric Populations *Michael B Higginbotham, MD*
☐	AARC95-050	Mechanical Limitations During Exercise *Charles G Irvin, PhD*
☐	AARC95-051	Utilizing the AARC Clinical Practice Guidelines *Garry W Kauffman, MPA, RRT*
☐	AARC95-052	Building a Collaborative Practice Team *Dorothy M Kite-Powell, CCRN, MSN*
☐	AARC95-053	Benchmarking Your Standards of Care *Michael Boroch MBA, RRT*
☐	AARC95-054	Building Your Outcome Management Goals *Michael Boroch, MBA, RRT*
☐	AARC95-055	Case Presentation--Outcomes Management *John S Sabo, MS, RRT*
☐	AARC95-056	Meeting the Demands of a "Reformed" Health Care System *William G Hinkle, PhD*
☐	AARC95-057	The Development of Core Curricula in Allied Health Programs *Leonard J Finocchio, MPH*
☐	AARC95-058	First Year Report on the Respiratory Care Accreditation Board (RCAB) *Thomas F Anderson, PhD, RRT*

Monday, December 4, 1995

☐	AARC95-061	Respiratory Care at the Crossroads: Evolution or Extinction? *Dean R Hess, PhD, RRT*
☐	AARC95-062	The Impact of Restructuring on Respiratory Care Education *Crystal L. Dunlevy, EdD, RRT*
☐	AARC95-063	Alternative Strategies in Clinical Education *Crystal L Dunlevy, EdD, RRT*
☐	AARC95-064	Enhanced Clinical Education in the Respiratory Care Lab *William F Galvin, MSEd, RRT, CPFT*
☐	AARC95-066	Pediatric Pulmonary Diagnostics: Aren't They Just Little Adults? *Susan Blonshine, BS, RRT, RPFT*
☐	AARC95-067	Do All Pediatric Tests Need To Comply with the ATS Standards? *Jack Wanger, MBA, RRT, RPFT*
☐	AARC95-068	Pediatric Challenges *Carl Montram, RRT, RPFT*
☐	AARC95-069	Assessment of Respiratory Control in the Fullterm Infant *Deborah K White, BS, RRT, RPFT*
☐	AARC95-070	Scientific Rationale for Clinical Use of Nitric Oxide *John E. Thompson, RRT*
☐	AARC95-071	Current and Future Applications of Nitric Oxide *Robert M Kacmarek, PhD, RRT*
☐	AARC95-072	Watch It!--Practical Aspects of Nitric Oxide *John E. Thompson, RRT*
☐	AARC95-073/74	The View of the Neonatologist; The View of the Pediatrician *Sherry E. Courtney, MS, MD; David E. Geller, MD*
☐	AARC95-075/76	The View of the Anesthesiologist; The View of the Pulmonologist *Charles, G Durbin Jr , MD; Richard Sheldon, MD*
☐	AARC95-077/78	The View of the Surgeon; The View of the Intensivist *Jay A Johannigman, MD; James M Hurst, MD*
☐	AARC95-081	Respiratory Muscle Retraining--Does It Improve Function? *Barry Make, MD*
☐	AARC95-082	Metabolic Testing--Does It Change the Exercise Prescription? *Mark W Millard, MD*
☐	AARC95-083	Five Important Articles You Should Have Read on Pulmonary Rehabilitation in 1995 *Barry Make, MD*
☐	AARC95-084	Pre-Hospital Management and Transport of the Trauma Patient *Jay A Johannigman, MD*
☐	AARC95-085	Management of the Pediatric Trauma Patient *Joseph J Tepas III, MD*

- ☐ AARC95-086 Management of the Pregnant Trauma Patient *David F Goldsberry, MD*
- ☐ AARC95-087 Management of the Burn Patient *C Wayne Cruse, MD*
- ☐ AARC95-088 Management of the Elderly, Injured Patient *James M Hurst, MD*
- ☐ AARC95-090 Meeting the Health Care Challenges of the Aging Population *Carl P Wiezalis MS, RRT*
- ☐ AARC95-091 Geriatric Nutritional Assessment *Pam Z Cacchione, RN MSN C-GNP*
- ☐ AARC95-092 Enhancing Communication and Teaching Techniques for the Older Patient *Pam Z Cacchione, RN, MSN, C-GNP*
- ☐ AARC95-093 Managing End-Of-Life Issues *Patrick J Cacchione*
- ☐ AARC95-094 Physiology of Alpha 1-Antitrypsin and Pathophysiology of Deficiency States *Michael J Krowka MD*
- ☐ AARC95-095 Clinical Features of Alpha 1-Antitrypsin Deficiency *James K Stoller, MD*
- ☐ AARC95-096 Current and Future Treatment Options for Alpha 1-Antitrypsin Deficiency: Augmentation Therapy, Gene Therapy *Charlie Strange, MD*
- ☐ AARC95-097 Lung Transplantation for Alpha 1-Antitrypsin Deficiency Emphysema *Marshall I Hertz, MD*
- ☐ AARC95-098 The 1995 JCAHO Standards -- The First Year's Experience *Kathleen Brinton, RRT*
- ☐ AARC95-099 Developing and Using an Effective Plan of Care *Vianna Zimbel, BS, CRTT*
- ☐ AARC95-100a Improving Organization Performance & Patient Satisfaction *Vianna Zimbel, BS, CRTT*
- ☐ AARC95-100b Questions and Answers -- Home Care The Impact of Accreditation

Tuesday, December 5, 1995

- ☐ AARC95-103 Equipment Validation in the Pulmonary Lab *Jack Wanger, MBA RRT, RPFT*
- ☐ AARC95-105 What Features Do We Really Need in Mechanical Ventilators? *Robert M Kacmarek, PhD, RRT*
- ☐ AARC95-106 The Consensus Statement from the 1995 Conference on Assessing Innovations on Mechanical Ventilatory Support *Thomas D East, PhD*
- ☐ AARC95-107 Defining Subacute Care Services: Patients, Settings, and Outcomes *Kathleen M Griffin, PhD*
- ☐ AARC95-108 Using Outcome Data to Justify Subacute Care Services *Dianne L Lewis, MS, RRT*
- ☐ AARC95-109 Providing Subacute Services in a Managed Care Environment *Kevin A Cormish, RRT*
- ☐ AARC95-110 Five Things I Learned this Year about Aerosol Therapy *Jon O Nilsestuen PhD, RRT*
- ☐ AARC95-111 Five Things I Learned this Year about CPR *Thomas A Barnes, EdD, RRT*
- ☐ AARC95-112 Five Things I Learned this Year about Managed Care *Michael Boroch, MBA, RRT*
- ☐ AARC95-113 Five Things I Learned this Year about Sleep Apnea *Karen J Stewart, BS, RRT*
- ☐ AARC95-114 Five Things I Learned this Year about What the RC Future Might Be (According to the Federal Government) *Cheryl A Brown, MHA*
- ☐ AARC95-115 Introduction and Overview: Identifying a Clinical Question and Formulating a Hypothesis *James K Stoller, MD, Shelley C Mishoe, PhD, RRT*
- ☐ AARC95-116/117 A Vignette: What is a Null Hypothesis?; Reviewing the Literature and Finding a Mentor *Dean R Hess, PhD, RRT, Joseph L Rau, PhD, RRT*
- ☐ AARC95-118-120 A Vignette: The p Value -- Just What Is It?; Validating Patient-Driven Protocols -- Choosing a Design; A Vignette: Getting Your Abstract Accepted *Dean R Hess, PhD, RRT, Charles G Durbin Jr, MD, Kaye Weber, MS, RRT*
- ☐ AARC95-121 Cystic Fibrosis: It's Not Just for Kids Anymore *Stanley B Fiel, MD*
- ☐ AARC95-122 Language of Managed Care *John R Walton, FACHE, RRT*
- ☐ AARC95-123 Competency Standard Development and Performance Management *John S Sabo, MS, RRT*
- ☐ AARC95-124 Preparing for Business Ventures *John R Walton, FACHE, RRT*
- ☐ AARC95-125 Outcomes Management: Paradigm Shift *Dorothy M Kite-Powell, RN, MSN, CCRN*
- ☐ AARC95-126 RCPs Should Insert Chest Tubes *Richard Sheldon, MD & Paul A Selecky, MD*
- ☐ AARC95-127 Waveforms and Graphics Should Be Monitored and Available on All Mechanically Ventilated Patients in the ICU *Robert S Campbell, RRT & Richard D Branson, RRT*
- ☐ AARC95-128 Hospitals Need RC Technical Directors *John S Sabo, MS, RRT & Michael Boroch, MBA, RRT*
- ☐ AARC95-129 Subacute Respiratory Care Services Can Effectively Be Delivered in an Acute Hospital Setting *Patrick J Dunne, MEd, RRT & Dianne L Lewis, MS, RRT*
- ☐ AARC95-130 Are RCPs Really Going To Be Able to Depend on Computers? *Thomas D East, PhD*
- ☐ AARC95-131 The Effects of State Licensure and Credentialing on Respiratory Home Care *Jill A Eicher, MPA*
- ☐ AARC95-132 Washington Update on Home Care *Cheryl A Brown, MHA*
- ☐ AARC95-135 Ethical Principles Relevant to the ICU *Gordon D Rubenfeld, MD*
- ☐ AARC95-136 Advance Directives and DNAR Orders *David J Pierson, MD*
- ☐ AARC95-137 The Nuts and Bolts of Withdrawing Life Support *Gordon D Rubenfeld, MD*
- ☐ AARC95-138 Cross-Training Isn't Just Diaper Changes and Feedings *John E Thompson, RRT*
- ☐ AARC95-139 Supplemental Oxygen Delivery for Pediatric Patients in Alternate Care Sites *Mark Wilson, MD*
- ☐ AARC95-140 Patient-Driven Protocols in the Perinatal-Pediatric Setting *John W Salyer, BS, RRT*
- ☐ AARC95-141 Neonatal or Pediatric Mechanical Ventilatory Management Outside the Hospital *William B Glomb, MD*
- ☐ AARC95-142 Airway Mechanics Laboratory Measurement *Robert A Brown, BS, RRT, RPFT*
- ☐ AARC95-144 Bronchial Provocation Testing Relationship to Airway Mechanics *Jack Wanger, MBA, RRT, RPFT*
- ☐ AARC95-145 Assessment of Airway Mechanics Outside the Pulmonary Function Laboratory *Charles G Irvin, PhD*

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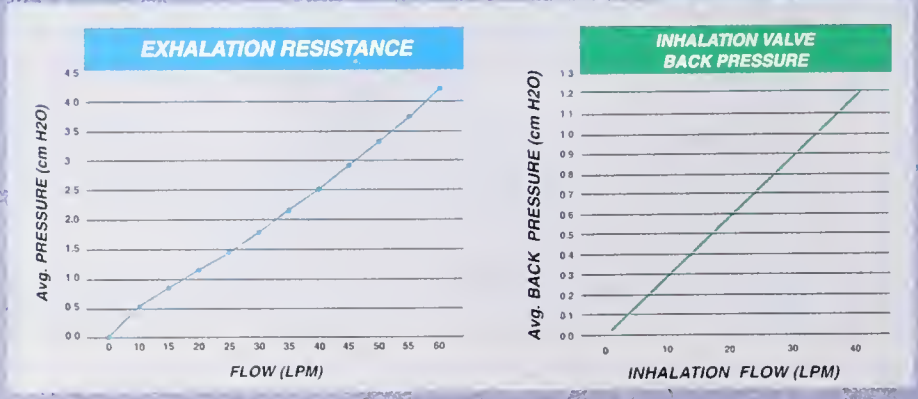

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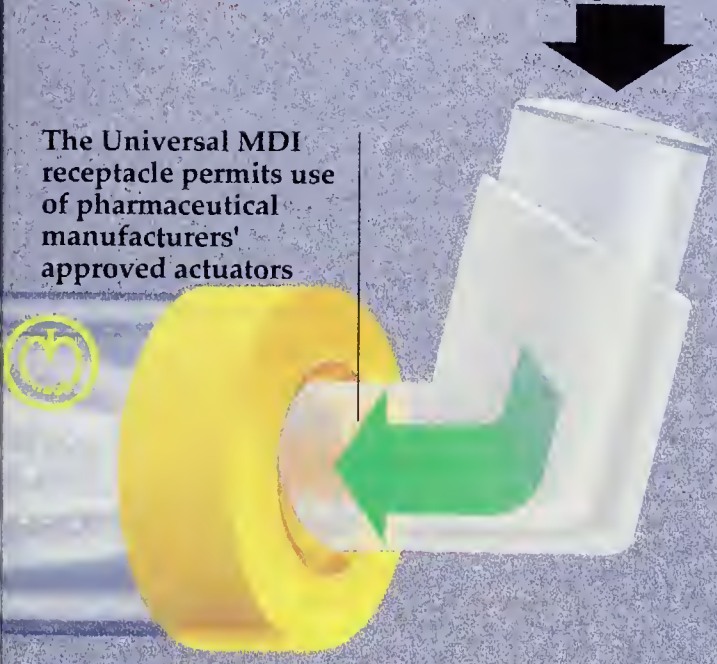

1. Drug delivery from holding chambers with attached facemaskML Everard et al, Archives of Disease in Children, Vol. 67, No. 5, May 1992

2. Substitution of Metered Dose Inhalers for Hand Held Nebulizers. Success and Cost Savings in a large, Acute Care Hospital, Bowton, DL, et al, Chest 101 (2) : 305-8, 1992 Feb.

3. Metered Dose Inhalers with Spacers vs Nebulizers for Bronchodilator Therapy in a Pediatric Emergency Department, Chou, KJ, et al, Am. J of Dis of Child. volume 147, Nn. 4 April 1993

4. Efficacy of Albuterol Administered by Nebulizer versus Spacer Device in Children with Accute Asthma, Karem, E., et al J. Ped 123 (2):313-7, 1993

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SUREMENTS & RESULTS: Hospital mortality for the 362 admissions was 24%. For the 167 patients aged 65 years or older, mortality was 30% at hospital discharge, 41% at 90 days, 47% at 180 days, and 59% at 1 year. Median survival for all patients was 224 days, and median survival for the patients who died within 1 year was 30.5 days. On multiple regression analysis, variables associated with hospital mortality included age, severity of respiratory and nonrespiratory organ system dysfunction, and hospital length of stay before ICU admission. Development of nonrespiratory organ system dysfunction was the major predictor of hospital mortality (60% of total explanatory power) and 180-day outcomes (54% of explanatory power). Respiratory physiological variables (respiratory rate, serum pH, P_{aCO_2}, P_{aO_2}, and alveolar-arterial difference in partial pressure of oxygen [$P_{AO_2}-P_{aO_2}$]) indicative of advanced dysfunction were more strongly associated with 180-day mortality rates (22% of explanatory power) than hospital death rates (4% of explanatory power). After controlling for severity of illness, mechanical ventilation at ICU admission was not associated with either hospital mortality or subsequent survival. CONCLUSIONS: Patients with COPD admitted to an ICU for an acute exacerbation have a substantial hospital mortality (24%). For patients aged 65 years or older, mortality doubles in 1 year from 30% to 59%. Hospital and longer-term mortality is closely associated with development of nonrespiratory organ system dysfunction; severity of the underlying respiratory function substantially influences mortality following hospital discharge. The need for mechanical ventilation at ICU admission did not influence either short- or long-term outcomes. Physicians should be aware of these relationships when making treatment decisions or evaluating new therapies.

Low-Dose Nebulized Morphine Does Not Improve Exercise in Interstitial Lung Disease—AO Harris-Eze, G Sridhar, RE Clemens, TA Zintel, CG Gallagher, DD Marciniuk. Am J Respir Crit Care Med 1995;152:1940.

Recent reports have suggested that low-dose nebulized morphine may improve exercise tolerance in patients with interstitial lung disease (ILD) by acting on peripheral opioid-sensitive pulmonary receptors. We therefore examined whether the administration of low-dose nebulized morphine would influence dyspnea or the breathing pattern during exercise of subjects with ILD and improve their exercise performance. Each of 6 subjects with ILD underwent 3 maximal incremental cycle ergometer tests, each test separated from the last by at least 3 days. Each exercise test was similar except that 30 minutes before exercise, the subjects received nebulized saline (control), morphine 2.5 mg, or morphine 5.0 mg, respectively, in double-blinded fashion. No significant differences were noted in exercise duration, maximal workload, or sense of dyspnea at the end of exercise in the control test and the tests with either morphine 2.5 mg or morphine 5.0 mg. Nor were significant differences noted in resting, submaximal, or end-exercise measurements of oxygen uptake ($\dot{V}_{O_2}$), carbon dioxide output ($\dot{V}_{CO_2}$), end-tidal CO_2 (P_{ETCO_2}), oxygen saturation (S_{aO_2}), minute ventilation ($\dot{V}_I$), respiratory frequency (f), tidal volume (V_T), or heart rate (HR) in the 3 tests. Low-dose nebulized morphine did not alter the subjects' breathing patterns or affect the relationship between dyspnea and ventilation during exercise. No significant side effects were noted. The administration of low-dose nebulized morphine to subjects with ILD neither relieves their dyspnea during exercise nor improves their maximal exercise performance.

Sleep-Disordered Breathing in African-American Elderly—S Ancoli-Israel, MR Klauber, C Stepnowsky, E Estline, A Chinn, R Fell. Am J Respir Crit Care Med 1995;152:1946.

Although sleep-disordered breathing (SDB) has been shown to be very prevalent in the elderly, little has been done to examine differences between the elderly of different racial groups. It has been well documented that SDB often results in hypertension and that hypertension is more common in African-Americans than in Caucasians. Therefore, one might suspect that SDB might be more common in African-Americans. Caucasians (n = 346) and African-Americans (n = 54) older than 65 years of age were studied. African-Americans reported less satisfaction with sleep ($p = 0.017$), more difficulty falling asleep ($p < 0.001$), more daytime sleepiness ($p = 0.0014$), and more frequent morning headaches ($p = 0.0043$). African-Americans napped 0.8 times more frequently per evening ($p = 0.05$) and 11 minutes longer per nap ($p = 0.019$) than did Caucasians, and they showed a trend toward more total sleep time (428 vs 408 min). Of greater interest was the fact that more African-Americans had severe SDB with a relative risk twofold as great (relative risk = 2.13) as that for Caucasians, which was confirmed in a logistic regression analysis where race was associated with the presence of SDB (RDI ≥ 30) independent of age, sex, and body-mass index. The mean RDI for those African-Americans with severe SDB was significantly higher than that for Caucasians (72.1 vs 43.3; $p = 0.014$).

The Impact of Exercise Reconditioning on Breathlessness in Severe Chronic Airflow Limitation—DE O'Donnell, M McGuire, L Samis, KA Webb. Am J Respir Crit Care Med 1995; 152:2005.

Exercise in chronic airflow limitation (CAL) is often limited by symptoms before the physiologic boundaries of maximal ventilatory or cardiovascular capacities are attained. Symptom amelioration should translate directly into improved exercise performance. We studied the impact of a 6-week supervised multimodality endurance exercise program (EXT) on perceived breathlessness (B) and leg effort (LE) and sought a physiologic rationale for symptom improvement. Thirty patients with CAL ($FEV_1/FVC = 42 \pm 2\%$, mean ± SEM) were tested before and after EXT. Their responses were compared with those of a matched control group (n = 30; $FEV_1/FVC = 44 \pm 2\%$) after a nonintervention period. Testing included pulmonary function tests, chronic dyspnea evaluation (Baseline/Transition Dyspnea Index [BDI/TDI]), and graded cycle exercise with cardioventilatory monitoring and Borg scaling of B and LE. Spirometry did not change (Δ) post-EXT. EXT significantly ($p < 0.001$) reduced chronic breathlessness ($TDI = +2.8 \pm 0.3$) compared with control ($TDI = 0.0 \pm 0.3$). Exertional symptoms of B and LE also fell ($p < 0.01$) after EXT (slopes of B and LE relative to $\dot{V}_{O_2}$ fell by 14 and 23%, respectively; $\Delta B/\dot{V}_{O_2}$ was associated with $\Delta LE/\dot{V}_{O_2}$, $r = 0.52$, $p < 0.01$). Post-EXT slopes of B over ventilation ($\dot{V}_E$) also decreased by 10% ($p < 0.025$). Total cycle work increased 142 ± 70% ($p < 0.001$) post-EXT and correlated primarily with $\Delta B/\dot{V}_{O_2}$ ($r = -0.64$, $p < 0.001$). The best correlate of $\Delta B/\dot{V}_{O_2}$ was $\Delta\dot{V}_E/\dot{V}_{O_2}$ ($r = 0.47$, $p < 0.001$). $\dot{V}_E$ fell in proportion to $\dot{V}_{CO_2}$ at a standardized work rate ($r = 0.91$, $p < 0.001$), whereas efficiency improved ($\dot{V}_{O_2}$/work rate slopes fell 12%, $p < 0.01$). In conclusion, supervised EXT effectively relieved both chronic and acute activity-related breathlessness in CAL, and thereby improved exercise tolerance. Relief of breathlessness was related to a fall in ventilatory demand during exercise as a result of enhanced mechanical efficiency. Reduced B for a given $\dot{V}_E$ after EXT may occur in part as a result of increased tolerance to the sensory perturbations that induce breathlessness in CAL.

Beneficial Effects of the "Open Lung Approach" with Low Distending Pressures in Acute Respiratory Distress Syndrome: A Prospective Randomized Study on Mechanical Ventilation—MBP Amato, CSV Barbas, DM Medeiros, GDP Schettino, GL Filho, RA Kairalla, D Deheinzelin, C Morais, ED Fernandes, TY Takagaki, and CRR De Carvalho. Am J Respir Crit Care Med 1995;152(6):1835.

Alveolar overdistention and cyclic reopening of collapsed alveoli have been implicated in the lung damage found in animals submitted to artificial ventilation. To test whether these phenomena are impairing the recovery of patients with acute respiratory distress syndrome (ARDS) submitted to conventional mechanical ventilation (MV), we evaluated the impact of a new ventilatory strategy directed at minimizing "cyclic parenchymal stretch." After receiving pre-established levels of hemodynamic, infectious, and general care, 28 patients with early ARDS were randomly assigned to receive either MV based on a new approach (NA, consisting of maintenance of end-expiratory pressures above the lower inflection point of the

$P \times V$ curve, $V_T < 6$ mL/kg, peak pressures < 40 cm H_2O, permissive hypercapnia, and stepwise utilization of pressure-limited modes) or a conventional approach (C = conventional volume-cycled ventilation, $V_T = 12$ mL/kg, minimum PEEP guided by F_{IO_2} and hemodynamics and normal P_{aCO_2} levels). Fifteen patients were selected to receive NA, exhibiting a better evolution of the P_{aO_2}/F_{IO_2} ratio ($p < 0.0001$) and of compliance ($p = 0.0018$), requiring shorter periods under $F_{IO_2} > 50\%$ ($p = 0.001$) and a lower F_{IO_2} at the day of death ($p = 0.0002$). After correcting for baseline imbalances in APACHE II, we observed a higher weaning rate in NA ($p = 0.014$) but not a significantly improved survival (overall mortality: 5/15 in NA vs 7/13 in C, $p = 0.45$). We concluded that the NA ventilatory strategy can markedly improve the lung function in patients with ARDS, increasing the chances of early weaning and lung recovery during mechanical ventilation.

Inhaled and Oral Corticosteroids: Their Effects on Bone Mineral Density in Older Adults—JF Marystone, EL Barrett-Connor, DJ Morton. Am J Public Health. 1995;85(12):1693.

Use of oral and inhaled corticosteroids and bone mineral density were examined cross-sectionally in 1673 community-dwelling white subjects aged 56 to 91 years. Bone mineral densities at the ultradistal and midshaft radii, hip, and lumbar spine were compared in users of inhaled (n = 34) and oral (n = 44) corticosteroids and nonusers. Women who used oral corticosteroids had significantly lower bone mineral densities at the midshaft radius, hip, and spine than never users. Women who used inhaled corticosteroids had bone mineral densities at the ultradistal radius, hip, and spine that were intermediate between those of oral corticosteroid users and those of never users. Bone mineral density did not vary significantly according to corticosteroid use in men.

Muscle Strength, Symptom Intensity, and Exercise Capacity in Patients with Cardiorespiratory Disorders—AL Hamilton, KJ Killian, E Summers, NL Jones. Am J Respir Crit Care Med 1995;152:2021.

The contribution of muscle strength to symptom intensity and work capacity was examined in normal individuals and patients with cardiorespiratory disorders. Respiratory muscle strengths (maximal inspiratory and expiratory pressures) and peripheral muscle strengths (leg extension, leg flexion, seated bench press, and seated row) were measured in 4,617 subjects referred for clinical exercise testing. Subjects then rated the intensity of leg effort, discomfort with breathing (dyspnea), and chest pain (Borg scale) during an incremental exercise task (100 kp · m · min^{-1} each minute) to capacity on a cycle ergometer. Subjects were classified into groups on the basis of pulmonary function, drug therapy for cardiac disorders, and the presence of chest pain during exercise with electrocardiographic changes indicative of myocardial ischemia. Respiratory and peripheral muscle strengths, normalized for differences in age, sex, and height, were significantly reduced in patients with cardiorespiratory disorders compared with normal individuals. Muscle strength was a significant contributor to symptom intensity and work capacity in both health and disease; a 2-fold increase in muscle strength was associated with a 25 to 30% decrease in the intensity of both leg effort and dyspnea and a 1.4- to 1.6-fold increase in work capacity. These results emphasize the need for an integrative approach in the assessment and therapeutic management of exercise intolerance, which considers the contribution of muscle weakness to excessive symptoms and reduced work capacity, in addition to the contribution of ventilatory, gas exchange, and circulatory impairments.

Passive Smoking and Impaired Endothelium-Dependent Arterial Dilatation in Healthy Young Adults—DS Celermajer, MR Adams, P Clarkson, J Robinson, R McCredie, A Donald, JE Deanfield. N Engl J Med 1996;334:150.

BACKGROUND: Passive smoking has been linked to an increased risk of dying from atherosclerotic heart disease. Since endothelial dysfunction is an early feature of atherogenesis and occurs in young adults who actively smoke cigarettes, we hypothesized that passive smoking might also be associated with endothelial damage in healthy young-adult nonsmokers. METHODS: We studied 78 healthy subjects (39 male and 39 female) 15 to 30 years of age (mean ± SD, 22 ± 4): 26 control subjects who had never smoked or had regular exposure to environmental tobacco smoke, 26 who had never smoked but had been exposed to environmental tobacco smoke for at least 1 hour daily for 3 or more years, and 26 active smokers. Using ultrasonography, we measured the brachial-artery diameter under base-line conditions, during reactive hyperemia (with flow increase causing endothelium-dependent dilatation), and after sublingual administration of nitroglycerin (an endothelium-independent dilator). RESULTS: Flow-mediated dilatation was observed in all control subjects (8.2 ± 3.1%; range, 2.1 to 16.7) but was significantly impaired in the passive smokers (3.1 ± 2.7%; range, 0 to 9; $p < 0.001$ for the comparison with the controls) and in the active smokers (4.4 ± 3.1% range, 0 to 10; $p < 0.001$ for the comparison with the controls; $p = 0.48$ for the comparison with the passive smokers). In the passive smokers, there was an inverse relation between the intensity of exposure to tobacco smoke and flow-mediated dilatation ($r = -0.67$, $p < 0.001$). In contrast, dilatation induced by nitroglycerin was similar in all groups. CONCLUSIONS: Passive smoking is associated with dose-related impairment of endothelium-dependent dilatation in healthy young adults, suggesting early arterial damage.

Oxygen Cost of Breathing in Patients with Emphysema or Chronic Bronchitis in Acute Respiratory Failure—V Jounieaux, J Mayeux. Am J Respir Crit Care Med 1995;152:2181.

This study compared the oxygen cost of breathing ($\dot{V}_{O_2}$ resp) in 19 patients with severe chronic obstructive pulmonary disease intubated for acute respiratory failure. Ten patients showed radiologic (X-ray and/or computed tomographic scan) evidence of emphysema. The remaining ones were considered as having chronic bronchitis. Measurements were made just before extubation. Despite similar expiratory airflow obstruction, patients with emphysema exhibited significantly higher $\dot{V}_{O_2}$ resp than patients with chronic bronchitis (109 ± 61 vs 42 ± 26 mL/min/m^2, respectively; $p < 0.006$). Moreover, emphysema was associated with nutritional depletion assessed through decreases in body-mass index (emphysema: 17.9 ± 3.5 kg/m^2; chronic bronchitis: 28.8 ± 8.2 kg/m^2; $p < 0.005$). This seemed to affect somatic stores (significant decreases in arm muscular circumference and triceps skinfold thickness, whereas visceral stores were preserved (no decreases in serum albumin, serum pre-albumin, and retinol binding protein). Malnutrition appeared to be the consequence of a hypermetabolic state of the respiratory muscles, with a significant negative correlation between $\dot{V}_{O_2}$ resp and body-mass index, arm muscular circumference, and triceps skinfold thickness ($p < 0.05$). Total oxygen consumption normalized for body surface was similar in the 2 groups. Thus, in emphysematous patients, the oxygen available for tissues other than respiratory muscles was significantly reduced (emphysema: 124 ± 51 mL/min/m^2; chronic bronchitis: 207 ± 78 mL/min/m^2; $p < 0.02$). This could explain nutritional differences observed between patients with emphysema and those with chronic bronchitis.

Prognosis and Outcomes of Patients with Community-Acquired Pneumonia: A Meta-Analysis—MJ Fine, MA Smith, CA Carson, SS Mutha, SS Sankey, LA Weissfeld, WN Kapoor. JAMA 1995;274(2):134.

OBJECTIVE: To systematically review the medical literature on the prognosis and outcomes of patients with community-acquired pneumonia (CAP). DATA SOURCES: A MEDLINE literature search of English-language articles involving human subjects and manual reviews of article bibliographies were used to identify studies of prognosis in CAP. STUDY SELECTION: Review of 4,573 citations revealed 122 articles (127 unique study cohorts) that reported medical outcomes in adults with CAP. DATA EXTRACTION: Qualitative assessments of studies' patient populations, designs, and patient outcomes were performed. Summary univariate odds ratios (ORs) and rate differences (RDs) and their associated 95% con-

fidence intervals (CIs) were computed to estimate a summary effect size for the association of prognostic factors and mortality. DATA SYNTHESIS: The overall mortality for the 33,148 patients in all 127 study cohorts was 13.7%, ranging from 5.1% for the 2,097 hospitalized and ambulatory patients (in 6 study cohorts) to 36.5% for the 788 intensive care unit patients (in 13 cohorts). Mortality varied by pneumonia etiology, ranging from less than 2% to greater than 30%. Eleven prognostic factors were significantly associated with mortality using both summary ORs and RDs: male sex (OR = 1.3; 95% CI, 1.2 to 1.4), pleuritic chest pain (OR = 0.5; 95% CI, 0.3 to 0.8), hypothermia (OR = 5.0; 95% CI, 2.4 to 10.4), systolic hypotension (OR = 4.8; 95% CI, 2.8 to 8.3), tachypnea (OR = 2.9; 95% CI, 1.7 to 4.9), diabetes mellitus (OR = 1.3; 95% CI, 1.1 to 1.5), neoplastic disease (OR = 2.8; 95% CI, 2.4 to 3.1), neurologic disease (OR = 4.6; 95% CI, 2.3 to 8.9), bacteremia (OR = 2.8; 95% CI, 2.3 to 3.6), leukopenia (OR = 2.5; 95% CI, 1.6 to 3.7), and multilobar radiographic pulmonary infiltrate (OR = 3.1; 95% CI, 1.9 to 5.1). Assessments of other clinically relevant medical outcomes such as morbid complications (41 cohorts), symptoms resolution (7 cohorts), return to work or usual activities (5 cohorts), or functional status (1 cohort) were infrequently performed. CONCLUSIONS: Mortality for patients hospitalized with CAP was high and was associated with characteristics of the study cohort, pneumonia etiology, and a variety of prognostic factors. Generalization of these findings to all patients with CAP should be made with caution because of insufficient published information on medical outcomes other than mortality in ambulatory patients.

Home Mechanical Ventilation Demographics and User Perspectives—RS Goldstein, JA Psek, EH Gort. Chest 1995;108(6):1581.

Although the number of users of home mechanical ventilation (HMV) is increasing, information regarding users' perceptions of the impact of HMV on their lives is incomplete. We administered an open-ended questionnaire to 98 (48 male, 50 female) HMV users aged (mean ± SD) 47.4 ± 19.5 years with COPD (9%), thoracic restrictive disease (43%), and neuromuscular disease (48%). At the time of survey, subjects had received HMV for 59.5 ± 58.3 months. Fifty-three percent were ventilated electively. Ventilator use was continuous (18%), at night only (37%), or at night with occasional daytime use (45%). Twenty-seven (28%) individuals identified themselves as being totally independent while 32 (33%) rated themselves as partially dependent on caregiver assistance for daily activites. Only 37 (38%) considered that they had made an informed choice when first starting HMV, whereas 54 (55%) did not. The impact of using a mechanical ventilator was believed to be overwhelmingly positive (87%). Positive comments regarding the impact of the ventilator on lifestyle were grouped in the following categories: life sustaining, facilitating mobility, and improving physical symptoms. Negative comments were grouped in the following categories: limiting mobility, equipment issues, and social implications. HMV users with a tracheostomy volunteered significantly fewer positive statements than those ventilated noninvasively ($p < 0.05$). Whereas 52 (53%) of users indicated that they had experienced initial difficulties in coping with the ventilator, only 11 (11%) identified difficulties at the time of the survey.

Noninvasive Nasal Mask Ventilation beyond the ICU for an Exacerbation of Chronic Respiratory Insufficiency—E Servera, M Pérez, J Marín, P Vergara, R Castaño. Chest 1995; 108(6):1572.

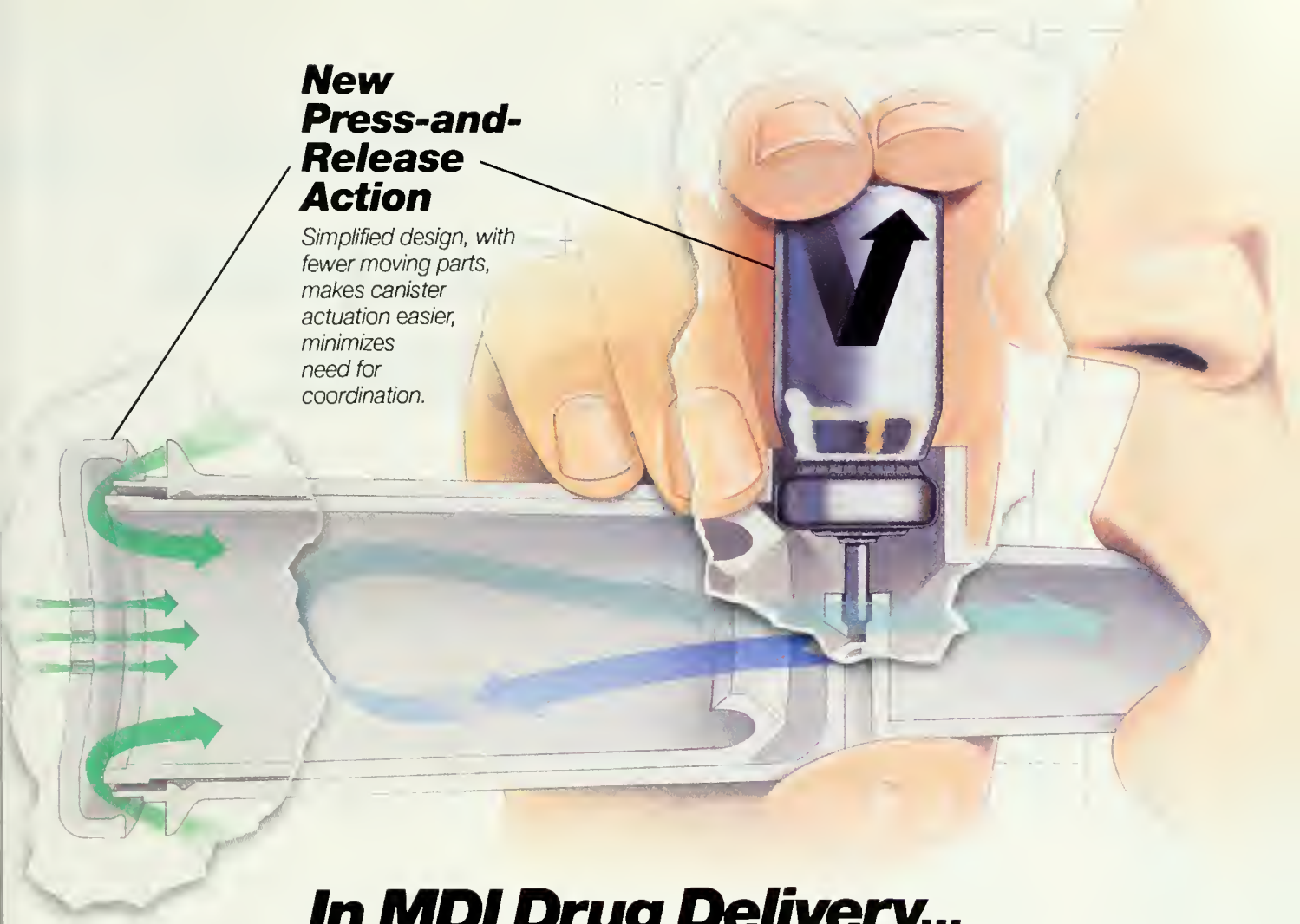
OBJECTIVE: To assess the usefulness of noninvasive nasal mask ventilation (NMV) in the treatment of an exacerbation of chronic respiratory insufficiency in patients stable enough to be admitted to a non-ICU ward. DESIGN: A prospective study in which the beneficial effect of NMV was compared with conservative treatment. SETTING: A ward of respiratory medicine of a tertiary referral teaching hospital. PATIENTS: The study group included 15 patients with acute respiratory acidosis. These patients had pH less than 7.35 and P_{aCO_2} more than 60 mm Hg, respiratory rate of 30 breaths or less per minute, hemodynamic stability, and alertness and willingness of cooperation with the NMV treatment. The control group consisted of 16 patients who fulfilled the same arterial blood gas requirements, retrospectively selected from the discharge forms of the ward of respiratory medicine for the year 1993. INTERVENTIONS: Patients underwent NMV for 2 sessions per day (1 in the morning and 1 in the afternoon), each session lasting 4 hours. A volumetric respirator (Monnal D; Taema; Paris, France) was used in 4 patients with restrictive disease. A positive-pressure ventilator (DP90; Taema; Paris, France) was used in 11 patients with obstructive disease. Control patients received standard medical, oxygen, and chest physical therapy. RESULTS: As compared with pre-NMV values, mean pH was significantly higher at 4 hours of NMV after the patient's ventilatory adaptation ($t = 8.814$, $p < 0.001$) and at the end of NMV ($t = 12.06$, $p < 0.001$). Ventilatory support also produced a significant improvement in hypercapnia (pre-NMV vs NMV after the patient's ventilatory adaptation, $t = 6.675$, $p < 0.001$; pre-NMV; vs post-NMV, $t = 6.976$, $p < 0.001$). Posttreatment pH and P_{aCO_2} values were significantly higher and lower, respectively, in NMV-treated patients than in controls. At the end of treatment, a significantly higher P_{aO_2}/F_{IO_2} ratio was documented in the study group than in controls (post-NMV vs posttreatment, $t = 2.846$, $p < 0.01$). CONCLUSIONS: NMV associated with standard treatment may be more beneficial than conservative treatment alone for improving blood gas exchange in patients with chronic respiratory insufficiency admitted to the hospital (but not the ICU) for an episode of acute decompensation and respiratory acidosis. *See the related editorial:* ***Technology Assessment.*** *DM Goodenberger. Chest 1995;108(6):1487-1488.*

Inhaled Budesonide Therapy for Patients with Stable COPD—P Weiner, M Weiner, Y Azgad, D Zamir. Chest 1995;108(6):1568.

A significant minority of patients with COPD have favorable response to corticosteroid treatment. In addition, the benefit of corticosteroid treatment may be outweighed by the side effects. Long-term administration of inhaled steroids is a safe means of treatment. We hypothesized that treatment with high-dose inhaled budesonide would improve clinical symptoms and pulmonary function in subjects with COPD, and that the response to inhaled β_2-agonist will serve to individualize steroid responders. We compared a 6-week course of 800 μg/d inhaled budesonide with placebo, separated by 4 weeks when no medication was taken, in a double-blind crossover trial, in 8 patients responding to inhaled β_2-agonist, and in 22 nonresponders with stable COPD. In 6 of 8 "responders to β_2-agonist," there was a significant improvement in the FEV_1 (defined as ≥ 20%) following inhaled budesonide, as compared with placebo. In the 22 "nonresponders to β_2-agonist," there was no significant improvement in the mean FEV_1 (1.41 ± 0.1 L before, and 1.61 ± 0.1 L after treatment) with inhaled budesonide or placebo. Over the 6-week course of treatment by either budesonide or placebo, the nonresponders reported similar β_2-agonist consumption (4.8 ± 0.2 and 5.0 ± 0.1 puffs per patient per day, respectively). However, there was a significant difference between the 2 periods of treatment in the responders as for the mean daily number of β_2-agonist inhalations (2.4 ± 0.1 in the budesonide period as compared with 5.3 ± 0.1 in the placebo period; $p < 0.005$). We conclude that treatment with inhaled steroids improved spirometry data and inhaled β_2-agonist consumption in about 25% of patients with stable COPD, and this rate is increased to about 75% in patients who respond to β_2-agonist inhalation. *See the related editorial:* ***Inhaled Corticosteroids in COPD—A Light at the End of the Tunnel?*** *TS deGuia. Chest 1995;108(6):1486-1487.*

The Effect of Late-Onset Ventilator-Associated Pneumonia in Determining Patient Mortality—MH Kollef, P Silver, DM Murphy, E Trovillion. Chest 1995;108(6):1655.

OBJECTIVE: To determine whether the development of late-onset ventilator-associated pneumonia (VAP) is associated with an increased risk of hospital mortality. DESIGN: Prospective cohort study. SETTING: ICUs of 2 university-affiliated teaching hospitals. PATIENTS: Three-hundred-fourteen patients admitted to an ICU who required mechanical ventilation for greater than 5 days. INTERVENTIONS: Prospective patient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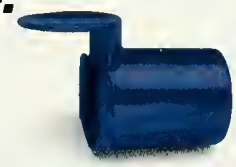

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surveillance and data collection. MEASUREMENTS: The primary outcome measures were the development of late-onset VAP (ie, occurring > 96 hours after intubation) and hospital mortality. RESULTS: Late-onset VAP was observed in 87 patients (27.7%). Thirty-four (39.1%) patients with late-onset VAP died during hospitalization compared with 85 patients (37.4%) without late-onset VAP (relative risk, 1.04; 95% confidence interval [CI], 0.76 to 1.43). Twenty patients (6.4%) developed late-onset VAP due to a "high-risk" pathogen (ie, *Pseudomonas aeruginosa*, Acinetobacter sp., *Xanthomas maltophilia*) with an associated mortality rate of 65%. Stepwise logistic regression analysis identified 5 variables as independent risk factors for hospital mortality ($p < 0.05$): an organ system failure index of 3 or greater (adjusted odds ratio [AOR], 3.4; 95% CI, 2.0 to 5.8; $p < 0.001$), having a nonsurgical diagnosis (AOR, 2.1; 95% CI, 1.3 to 3.6; $p = 0.002$), a premorbid lifestyle score of 2 or greater (AOR, 1.8; 95% CI, 1.1 to 2.9; $p = 0.015$), acquiring late-onset VAP due to a 'high-risk' pathogen (AOR, 3.4; 95% CI, 1.2 to 10.0; $p = 0.025$), and having received antacids or histamine type-2-receptor antagonists (AOR, 1.7; 95% CI, 1.0 to 2.9; $p = 0.034$). Additionally, we found the occurrence of late-onset VAP due to high-risk pathogens to be the most important predictor of hospital mortality among patients developing VAP (AOR, 5.4; 95% CI, 2.8 to 10.3; $p = 0.009$). CONCLUSIONS: Nosocomial pneumonia due to certain high-risk micro-organisms is an independent risk factor for hospital mortality among patients requiring prolonged mechanical ventilation. We suggest that future investigations of late-onset VAP stratify patient outcomes according to the distribution of high-risk pathogens when reporting their results.

Airway Protection during Experimental CPR—M Fukui, MH Weil, W Tang, L Yang, S Sun. Chest 1995;108(6):1663.

BACKGROUND: Experimental studies recently demonstrated that positive pressure ventilation may not be essential for initial cardiopulmonary resuscitation. Nevertheless, oxygen enrichment of inspired gas mixtures and spontaneous gasping were associated with increased resuscitability and survival after cardiac arrest. However, as yet unresolved is the benefit of early airway control under conditions simulating 'sudden death' due to ventricular fibrillation. METHODS: Twenty adult, male Sprague-Dawley rats were randomly assigned to 1 of 2 groups in which the airway was unprotected or protected by an oropharyngeal airway of our design. Cardiac arrest was induced by an alternating current delivered to the right ventricular endocardium. Oxygen was delivered to a hood that was loosely applied over the head of each animal at a flowrate of 1 L/min. Precordial compression was initiated after 4 minutes of untreated ventricular fibrillation and defibrillation was attempted 6 minutes later. After spontaneous circulation had been restored, a tracheostomy was performed and the animals were mechanically ventilated with 100% oxygen for an additional interval of 1 hour. Animals were then returned to their cages and observed for an additional 24 hours. RESULTS: Spontaneous circulation was restored in each of the animals who had an oropharyngeal airway and 9 of 10 animals in the absence of an artificial airway. In each group, 7 animals survived for more than 24 hours. Animals in which the airway had been protected had significantly greater frequency of spontaneous gasping (28 ± 13/min vs 13 ± 9/min; $p < 0.05$) and significantly higher arterial oxygen saturation ($77 \pm 19\%$ vs $55 \pm 25\%$; $p < 0.05$). CONCLUSION: In the setting of experimental cardiac resuscitation, the insertion of an artificial airway increased the frequency of spontaneous gasping and arterial oxygenation. Nevertheless, no significant differences in resuscitability or postresuscitation survival were associated with insertion of the artificial airway. *See the related editorial:* ***Is Mouth-to-Mouth Ventilation Necessary for Successful Resuscitation?*** *AH Idris. Chest 1995;108(6):1490-1491.*

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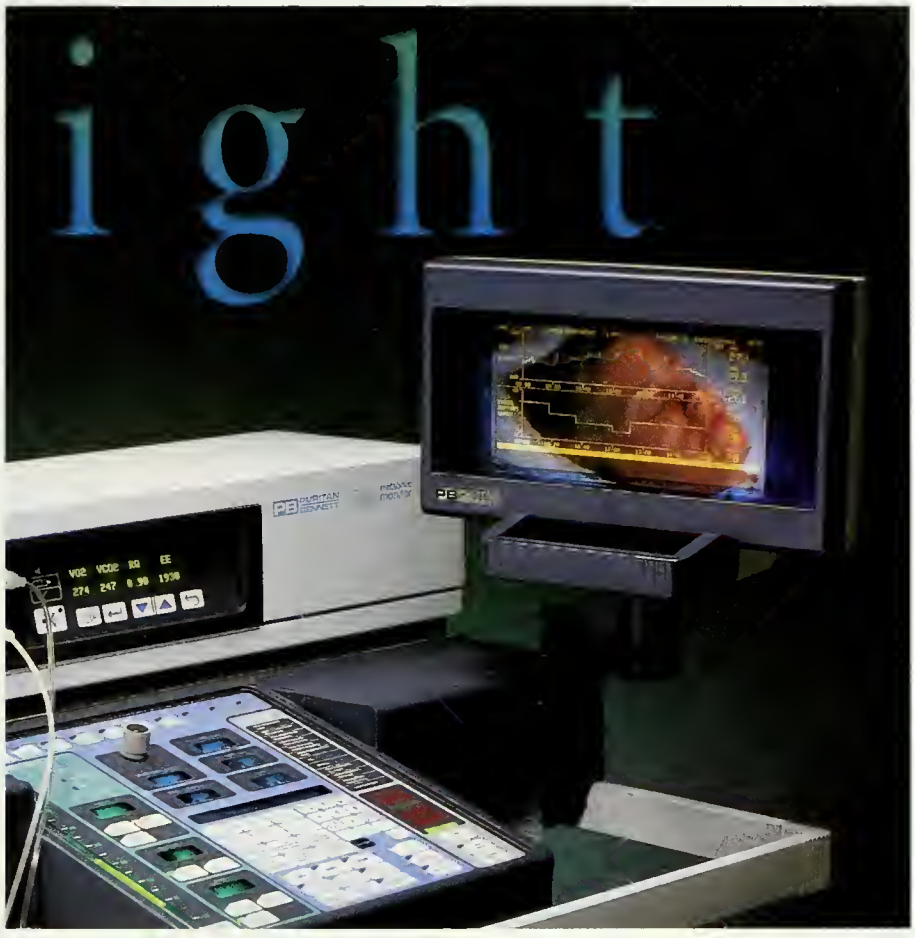

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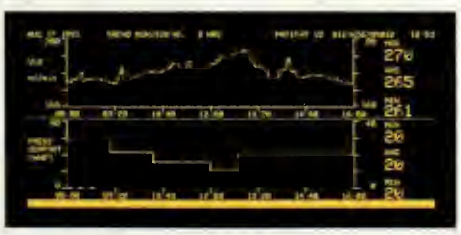

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New! Improved! or Nothing New Under the Sun?

Last autumn (1995), a report on treating acute exacerbations of chronic obstructive pulmonary disease (COPD) with noninvasive positive-pressure ventilation (NIPPV) led to many inquiries at our Respiratory Care Center about the availability of this 'new' treatment.[1] A few senior therapists and pulmonologists answered that it had been available for over 30 years![2] Perhaps this was a bit unfair because IPPB *had* improved as it evolved into NIPPV.[3] For example, the patient-ventilator interface is more comfortable, the pressure generator is more sophisticated, oxygen saturation (and other) monitors are available to increase safety, and NIPPV is used more selectively. What is new to general physicians is ancient history to respiratory care practitioners, of whom some fail to remember its many incremental improvements.

This month in RESPIRATORY CARE, Boix and colleagues[4] report their experience with a new noninvasive ventilator in patients with an acute exacerbation of COPD. They compared external high-frequency oscillatory ventilation (EHFOV) provided by a Hayek oscillator with modest amounts of nasal positive-pressure ventilation for brief periods in 20 patients with severe obstruction and ventilatory failure. The patients were carefully chosen: they received medical therapy for 1 or 2 days; none was acidotic; none required intubation; and all were able to stop supplemental oxygen during testing. The two ventilatory modes were equally effective in lowering P_{aCO_2}, yet respiratory rate paradoxically doubled with EHFOV (more about this later). Neither mode produced adverse cardiovascular effects. This report suggests that EHFOV is as effective as low-pressure NIPPV for treating ventilatory failure in selected patients with acute exacerbations of COPD. In the future, respiratory care practitioners may have a new therapy for this common problem.

This report of a new high-frequency negative-pressure ventilator is surprising for several reasons. First, the usual indication for high-frequency ventilation is diffuse alveolar disease in infants, not COPD in adults.[5] Second, the usual indication for negative-pressure ventilation is restrictive, not obstructive pulmonary disease.[6] Third, negative-pressure ventilation has been disappointing for treating *stable* COPD patients.[7] Surprise leads to curiosity and questions quickly arise. Some are listed as follows:

What is external high-frequency oscillatory ventilation? A cuirass ventilator applies sinusoidal pressure waves to the thorax and abdomen at higher than conventional ventilatory rates, eg, 60-2,400 cycles/min.[8] Tidal volumes are small and become smaller with higher rates. Oscillatory implies active inspiration (air is pulled-in) and expiration (air is pushed-out).[8]

How does external high-frequency oscillatory ventilation differ from other forms of high-frequency ventilation? High-frequency positive-pressure ventilators and jet ventilators deliver gas boluses to the airway opening; respiratory recoil empties the lung, ie, expiration is passive. High-frequency chest compressions from a pressure cuff literally squeeze air out of the lungs; respiratory recoil refills the lung, ie, inspiration is passive.[8-10]

What is the Hayek oscillator? A decade ago in the laboratory, Hayek and co-workers superimposed high-frequency oscillations from a piston pump on a baseline negative pressure from a vacuum pump and delivered the resulting oscillating negative pressure to a cuirass.[11] The baseline pressure preserved functional residual capacity (FRC) while the oscillations provided ventilation by expanding and compressing the chest and upper abdomen. The intent was to prevent the reduction of FRC observed with high-frequency chest compression devices.[9,10] In turn, this prevents airway closure, improves gas exchange, and augments ventilator effectiveness.

The clinical Hayek oscillator consists of a cuirass, power unit that contains the 2 pumps and control unit, a wide-bore hose for delivering the oscillating negative pressure to the cuirass, and a narrow-bore hose for monitoring extra-thoracic (cuirass or chamber) pressure. The operator sets frequency, inspiratory and expiratory pressures, and inspiratory-expiratory ratio; the control unit makes automatic adjustments.[12-15]

What are the operating characteristics of the Hayek oscillator? There is a paucity of reports in normal subjects.[12,13] The two evaluations in normal subjects rely on noninvasive tests such as end-tidal P_{CO_2}, pulse oximetry, impedance plethysmography, and pneumotachography. These tests are less reliable when used to assess high-frequency and cuirass ventilators.

The Hayek oscillator has two 'personalities.' At lower frequencies (15, 30 and 60 cycles/min), it behaves as a conventional ventilator, ie, the pressure span (difference between maximum inspiratory and expiratory pressures) directly affects tidal volume, minute ventilation, and gas exchange.[12,13] Average chamber pressure (analogous to mean airway pressure) has a small effect at 15 cycles/min.[13] At higher frequencies (120 and 180 cycles/min), the oscillator behaves as a high-frequency ventilator. With a relatively large pressure span of 30 cm H_2O, tidal volume imposed by the oscillator is approximately 200 mL (nearly anatomic dead-space volume), minute ventilation approaches 0 L/min, and spontaneous respiratory rate drops to about 6 breaths/min.[12,13]

Is the Hayek oscillator really a high-frequency ventilator? As noted, it works in normal subjects. The dramatic drop in respiratory rate seems to confirm the drop in end-tidal P_{CO_2}. Bradypnea and apnea have been observed with other high-frequency ventilators and may represent a reflex response to hypocapnia or chest-wall receptor stimulation.[16]

It also maintained adequate ventilation (by end-tidal P_{CO_2}) in patients undergoing tracheal surgery whose pulmonary status was not described.[14]

It improved ventilation (according to end-tidal CO_2) in stable COPD patients, half of whom were hypercapnic[15] and lowered P_{aCO_2} in unstable COPD patients.[4] It is possible that the increased respiratory rate observed by Boix and co-workers may indicate suboptimal application of EHFOV.[16]

Caveat lector. The study of Boix and co-workers provides an incremental increase in knowledge about the Hayek oscillator. However, there is much to learn about this new device, and it is important to keep the following in mind. First, Boix and co-workers selected their patients very carefully. Further study is needed in more acutely ill patients who may eventually require intubation. Second, EHFOV was used only briefly in this and other studies (≤ 45 minutes). It is not known whether patients will tolerate longer treatments, eg, 6 hours. Third, fixed ventilator settings were used in both studies involving COPD patients.[4,15] Acutely ill patients may benefit from bedside adjustments, eg, using a lower inspiratory-expiratory ratio or higher pressure span. Fourth, adverse effects were not seen because of careful patient selection. The potential remains for problems with overinflation[17] and diminished venous return.[18]

The combination of high-frequency and negative-pressure ventilation creates new opportunities. The ability to avoid the complications of intubation and positive-pressure ventilation justifies continued investigation. Augmented mucociliary clearance may be an added benefit.[19] If 'fine tuning' EHFOV produces bradypnea or apnea, then fatigued respiratory muscles may have a chance to rest.

This hybrid ventilator also creates challenges. Obtaining a good cuirass fit, monitoring adequacy of ventilation, and avoiding air trapping quickly come to mind.

Additional clinical evaluation of this new ventilator is required. Experienced practitioners may recall the excitement generated by high-frequency ventilators. We now realize that these ventilators have a fairly small "safe therapeutic window."[20]

Ultimately, prospective, controlled trials will determine whether the Hayek oscillator is truly New! Improved! and define its place in the respiratory armamentarium.

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Comparison of Nasal Positive-Pressure Ventilation to External High-Frequency Oscillatory Ventilation in Severe COPD

Jesús Héctor Boix MD, Manuel Tejeda MD, Faustino Álvarez MD, and Alfonso Bataller MD

BACKGROUND: Noninvasive mechanical ventilation is being used increasingly to obviate the necessity for endotracheal intubation and mechanical ventilation in patients in respiratory distress from acute exacerbations of chronic obstructive pulmonary disease (COPD). METHODS: The short-term (45-minute) therapeutic efficacy of nasal intermittent positive-pressure ventilation (NIPPV) and external high-frequency oscillatory ventilation was assessed in 20 COPD patients with an acute decompensation episode. Supplemental oxygen was discontinued and the two ventilatory methods were alternated. During the 24-48 hours after admission, the patient completed a 3-hour study session divided into four 45-minute periods, ie, baseline, first ventilatory mode, pause, and second ventilatory mode. NIPPV was delivered by a standard pressure ventilator in the assist mode, with a commercial nasal mask (Sullivan). The Hayek oscillator was used for high-frequency oscillatory ventilation. RESULTS: After 45 minutes of NIPPV, we observed statistically significant decreases in the respiratory rate and P_{aCO_2} and significant increases in S_{aO_2} and pH. After 45 minutes of high-frequency oscillatory ventilation, we observed statistically significant decreases in P_{aCO_2} and S_{aO_2} with significant increases in the respiratory rate and pH. Differences between decreases in P_{aCO_2} attained with both modes of ventilation were less than 5 torr in 13 patients, between 5 and 10 torr in 6 patients, and higher than 10 torr in 1 patient. CONCLUSIONS: Use of either method of ventilatory support resulted in improvement in alveolar ventilation, with significant changes in P_{aCO_2} and pH. Given that cooperation from the patient to decrease respiratory-muscle activity is required for successful NIPPV, the degree of the patient's cooperation is an important aspect to be considered in the selection of one of the two techniques. [Respir Care 1996;41(3):187-190]

Introduction

Respiratory muscle fatigue has been considered to be a contributory factor in the genesis of hypercapnic respiratory failure in chronic obstructive pulmonary disease (COPD).[1,2]

Invasive mechanical ventilation provides temporary relief to the respiratory muscles but exposes the patient to a variety of complications[3-5] resulting from the intubation procedure, during the course of ventilation, or after removal of the tube. Noninvasive methods of ventilation have been recognized as a means to improve gas exchange and to reduce respiratory-muscle work in obstructive lung disease.[6-8] Noninvasive intermittent positive-pressure ventilation (NIPPV) using a nose or face mask in appropriately selected patients can obviate endotracheal intubation and shorten intensive care unit (ICU) stay.[9-11] On the other hand, externally applied high-frequency oscillatory ventilation (EHFOV), which is a special form of negative-pressure ventilation, has been used effectively in short sessions in severe COPD and respiratory failure for assisting ventilation and relieving muscle fatigue.[12,13]

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None of the authors has a financial interest in any of the products mentioned in the text or in competing products.

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It was assumed that EHFOV, like NIPPV, could be used as a method of ventilatory support in patients with severe COPD. This study was conducted to compare the efficacy of NIPPV via nasal mask and EHFOV using the Hayek oscillator[14]* for ventilating patients with severe unstable COPD. With the Hayek oscillator, both inspiration and expiration are actively controlled.

Methods

Twenty patients [17 men and 3 women with a mean (SD) age of 63.3 (5.5) years] with severe COPD admitted to the two hospitals with which the authors are associated for treatment of an acute exacerbation episode comprised the convenience sample for the study. The patients fulfilled the American Thoracic Society criteria for COPD.[15] Acute decompensations were defined by increased cough, purulent sputum production, recent and rapid worsening of dyspnea with paradoxical abdominal motion or thoracoabdominal asynchronism, $P_{aO_2} < 60$ torr, $P_{aCO_2} > 50$ torr on admission when breathing room air, and/or severe pulmonary obstruction. Mean (SD) values for pulmonary function variables expressed as % predicted were forced vital capacity (FVC) 48.7 (12.8), forced expiratory volume in one second (FEV_1) 30.5 (5.2), FEV_1/FVC 42.1 (9.3), and forced expiratory flow after 50% of vital capacity has been expelled (FEF_{50}) 12 (3.5). Criteria for excluding patients from the study included need for endotracheal intubation and mechanical ventilation or a history of cardiovascular disease or evidence of pneumonia. Patients received standard treatment for COPD including low-flow oxygen therapy by nasal cannula, bronchodilator drugs (aminophylline, β_2-adrenoceptor agonists), corticosteroids, antibiotic therapy (amoxicillin with clavulanic acid), and chest physiotherapy. The study protocol was approved by the Ethics Committee of the participating hospitals.

Both noninvasive ventilatory modes were used at 24-48 hours after admission with the patient in a semirecumbent position, and after discontinuation of supplemental oxygen treatment. Each patient completed a 3-hour study session divided into four 45-minute periods, ie, baseline, first ventilatory mode, weaning of mechanical ventilation (pause), and second ventilatory mode. Patients in whom an odd number had been assigned when entering the study underwent EHFOV as the first ventilatory mode and then NIPPV; whereas, those with an even number had NIPPV followed by EHFOV. At the time of the study, the mean APACHE II score was 17.1 (2.3) and the mean gradation of dyspnea was 2.5 (0.7) according to Borg perceived dyspnea scale.[16]

The Hayek oscillator consists of a chest enclosure, a power unit, and a control unit. The chest enclosure (cuirass) is made of clear, flexible, lightweight plastic and is bordered by soft foam rubber. It is designed to fit snugly over the chest and upper abdomen. The cuirass is connected to the power unit with wide-bore tubing. Within the power unit is a diaphragmatic pump with a maximum stroke of 2 L, which can operate over a wide range of frequencies to generate an oscillating pressure. An additional vacuum pump enables this oscillation to be superimposed on a negative baseline. Ventilator settings were similar to those used in other studies[13,17] and included F_{IO_2} 0.21, frequency 120 cycles/minute, inspiratory pressure −30 cm H_2O, expiratory pressure +5 cm H_2O, I-E ratio 1:2. Both inspiratory and expiratory phases of respiration are actively controlled.

Intermittent positive-pressure ventilation was delivered by a standard ventilator used for the application of continuous positive airway pressure for the treatment of sleep apnea in the assisted mode, with a commercial nasal mask with 'air cushion.' The procedure of NIPPV was explained and the patient was made familiar with the nasal mask. Ventilator settings included F_{IO_2} 0.21 and an inspiratory positive airway pressure of 10 cm H_2O.[11,18]

Arterial blood was sampled via a catheter inserted in a radial artery using prepared heparinized syringes and analyzed immediately. Oxygen saturation was continuously monitored by pulse oximetry. Vital signs and blood gases (P_{aO_2}, P_{aCO_2}) were recorded at the end of each 45-minute period. Tolerance to each mode of ventilatory support was assessed on the basis of subjective criteria (acceptability of the method and adaptation to ventilation) and objective criteria (perceived dyspnea and complications).

Results are shown as means with standard deviations. For statistical comparisons we used the Wilcoxon test and the analysis of variance. A p value of < 0.05 was considered significant.

Results

Mean (SD) values of blood gases, pH, oxygen saturation, bicarbonate, base excess, heart rate, respiratory rate, and systolic and diastolic arterial pressure during the study period are shown in Table 1.

According to changes in P_{aCO_2}, the ventilatory efficacy of NIPPV and EHFOV was similar. Differences between decreases in P_{aCO_2} attained with both modes of ventilation were less than 5 torr in 13 patients, between 5 and 10 torr in 6 patients, and higher than 10 torr in 1 patient.

Acceptance of ventilatory support was excellent; although at the beginning of NIPPV, 15 patients had to be taught to adapt their inspiratory effort to the positive pressure and to avoid air leaks. At the end of the study, the dyspnea value decreased in 17 patients and remained unchanged in the remaining 3. No complications were observed during the study. All patients eventually recovered without requiring intubation.

*Suppliers are identified in the Product Sources section at the end of the text.

Table 1. Means and Standard Deviations of the Variables at Baseline and after 45 Minutes of External High-Frequency Oscillatory Ventilation (EHFOV), No Ventilation (Pause), and Nasal Intermittent Positive-Pressure Ventilation (NIPPV).

Variable	Baseline*	EHFOV	Pause	NIPPV
pH	7.37 (0.06)	7.40 (0.03)	7.38(10.07)	7.41 (0.05)†
P_{aCO_2} (torr)	64.4 (12.0)	56.4 (9.5)	65.4 (13.8)	56.5 (11.6)†
P_{aO_2} (torr)	52.9 (19.1)	51.1 (22.3)	52.0 (15.7)	52.5 (8.2)
Calculated S_{aO_2} (%)	81.72 (9.5)	80.0 (10.8) §	81.8 (8.97)	84.7 (6.98)†
HCO_3^- (mmol/L)	36.3 (4.3)	36.4 (3.8)	36.4 (4.4)	35.6 (4.7)
Base excess (mmol/L)	10.1 (3.8)	10.5 (3.5)	10.2 (3.7)	10.1 (3.9)
Heart rate (beats/min)	99.8 (13.7)	98.0 (14.2)	100.2 (13.8)	96.6 (14.4)
Respiratory rate (breaths/min)	30.3 (5.2)	58.6 (30.4)†§	31.0 (4.9)	19.6 (6.3)†
Systolic pressure (mm Hg)	123.4 (18.3)	126.4 (14.8)	121.9 (16)	122.2 (15.5)
Diastolic pressure (mm Hg)	71.3 (7.7)	74.8 (9.1)	72.5 (8.12)	71.7 (8.4)

*Differences between Baseline and Pause values are not statistically significant; † $p < 0.001$ compared to Baseline; ‡ $p < 0.05$ compared to Baseline; § $p < 0.001$ compared to NIPPV.

Discussion

Twenty patients with acute decompensations of COPD have been successfully ventilated noninvasively using NIPPV or EHFOV. Both modes of ventilatory support caused an improvement in alveolar ventilation, with significant changes in P_{aCO_2} and pH. As far as we are aware, this is one of the few reports of negative-pressure ventilation in unstable COPD. In addition, no study has directly compared NIPPV to EHFOV.

The good tolerance to both methods and absence of complications confirm the data reported in the literature[13,18-21] and appear to be important elements to be taken into consideration. However, although alertness and willingness to cooperate with the noninvasive ventilatory method were necessary to participate in the study, NIPPV was time-consuming for the health-care team and seems to require specific technical capacities and active cooperation by the patient.[22] This latter condition—not always present in all decompensated, severely hypercapnic COPD patients—may be an important aspect that may influence the selection of the most appropriate ventilatory support technique.

The initial results obtained with EHFOV in the management of respiratory failure due to COPD, particularly to prevent the need for intubation and mechanical ventilation, are encouraging.[13] In contrast to other modalities of external ventilation,[23,24] the Hayek oscillator controls both phases of respiration (ie, active inspiration and expiration), achieving high frequencies and higher volumes. In the study of Piquet et al,[12] high-frequency chest wall oscillation superimposed on spontaneous expiration in 12 male patients with severe COPD improved gas exchange and relieved the load of inspiratory muscles. According to these authors, this technique represents a new application of transthoracic high-frequency ventilation and can have a positive impact on physiotherapy and care of ambulatory patients with chronic air-flow obstruction. Spitzer and associates[13] studied the effect of the Hayek oscillator on 20 patients with severe COPD. The similar response of the patients to four different frequencies (60, 90, 120, and 140 cycles/min) indicates that frequencies above 140 cycles/minute would not be more effective than those tested. Although we used a fixed frequency of 120 cycles/minute, the frequency chosen may be adjusted in accordance with the comfort of the patient during application of the oscillator. On the other hand, amplitude should be selected individually on the basis of severity of disease and compliance of the chest wall. Lean patients require lower amplitude than obese patients, and patients with less impairment require lower amplitude than severely impaired patients.[13]

The increases in S_{aO_2} in our study should be interpreted with caution because oxygen saturation was calculated from arterial blood gas analysis. Further, our study was a short-term study.

It has been postulated that NIPPV improves ventilation and gas exchange by resting the fatigued muscles, increasing lung compliance, or resetting respiratory center sensitivity to CO_2. These theories, however, are not mutually exclusive and could contribute more or less depending on the patient.[8] The mechanical and physiologic changes responsible for the beneficial effects of EHFOV (in which spontaneous ventilation is overridden almost completely by the rapid action of the device) have not been fully explored, although it has been suggested that a reduction in P_{aCO_2} and improvement in respiratory function may be secondary to reversal of respiratory muscle fatigue.[12,13]

In summary, NIPPV and EHFOV appear to be valuable options in the treatment of patients with severe COPD, especially in the course of acute decompensations. Supplemental oxygen can be provided during NIPPV and EHFOV, if needed, to obtain an adequate arterial saturation. Adaptation to EHFOV is easy because active cooperation is not required. Therefore the degree of the patient's cooperation is an important feature that influences the choice of the method. Because NIPPV is not tolerated by some patients, EHFOV may be a useful alternative. In our opinion, both methods can be effi-

tively used in severe COPD and respiratory failure for assisting ventilation and reducing the needs for intubation and mechanical ventilation and for improving outcome in patients with acute exacerbations of COPD.

ACKNOWLEDGMENTS

We are indebted to Marta Pulido MD for editing the manuscript and translating it into English.

PRODUCT SOURCES

Hayek Oscillator:
Cardiomedic, FG/9300/9, Madrid, Spain
Possible U.S. Source: Plessy Medical Equipment (U.S.) Inc, Charlotte NC

Blood Gas Analyzer:
ABL 520, Radiometer America, Westlake OH

Pulse Oximeter:
Ohmeda 5250, Ohmeda, Liberty Corner NJ

Pressure-Limited Ventilator:
DP90, Taeme, Paris, France

Nasal Mask:
Sullivan, Possible U.S. Source: ResMed Corp, San Diego CA

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Prediction of the Resting Energy Expenditure of Mechanically Ventilated Patients in the Pediatric Intensive Care Unit

Mohamad F El-Khatib and Bonnie J Rosolowski RRT

INTRODUCTION: Critically ill infants and children who require mechanical ventilation are susceptible to malnutrition. Formulas for determining energy requirements in these patients have not been accurate and reliable. Knowledge of their energy requirements is essential for designing nutritional regimens to reduce carbon dioxide production. Inappropriate nutritional support may add to the effects of disease and prolong recovery and mechanical ventilation. OBJECTIVE: We attempted to derive new equations for estimation of measured energy expenditures (MEE) in children and infants receiving mechanical ventilation in the pediatric intensive care unit (PICU). METHODS: We retrospectively compared the values of MEE to those predicted by the Harris-Benedict (HB), the World Health Organization (WHO), and the Schofield equations, for all PICU patients evaluated with a metabolic cart from 1/1/92 to 3/31/95. Indirect calorimetry was performed on mechanically ventilated patients receiving oxygen concentrations ≤ 0.60 and with no audible air leak. RESULTS: 60 children (34 boys; 26 girls) with a mean (SD) age of 7 (6.8) years were identified, and 77 measurements were performed. MEE was significantly less than energy expenditure estimated by HB or WHO equations. The Schofield equation significantly underestimated MEE. Equations resulting from multiple linear regression analysis of MEE were: Boys: MEE (Kcal/day) = 3.3 Height (cm) + 5.7 Weight (kg) + 75.7 Age (years) + 42.3 (r^2 = 0.78); girls: MEE (Kcal/day) = 13.9 Height (cm) + 9.5 Weight (kg) − 42.2 Age (years) − 507 (r^2 = 0.85). CONCLUSION: Accurate, direct measurements of energy expenditure are recommended, but when a metabolic cart is not available, the equations derived from our data should provide a good estimation. [Respir Care 1996;41(3):191-196]

Introduction

Metabolic measurements for determination of oxygen consumption ($\dot{V}_{O_2}$), carbon dioxide production ($\dot{V}_{CO_2}$), respiratory quotient (RQ), and resting energy expenditure (REE) are essential to patient nutritional aid and assessment,[1-3] assessment of weaning success and outcome,[4,5] and assessment of the relationship between oxygen delivery ($\dot{D}_{O_2}$) and $\dot{V}_{O_2}$.[1,6] Malnutrition is a common problem in critically ill patients. Although few data are available to precisely define the incidence of malnutrition in mechanically ventilated patients, it occurs in as many as 25% of respiratory intensive-care-unit patients.[7,8] Although poor nutritional status does not appear to predispose to the need for mechanical ventilation, malnourished patients who require mechanical ventilation have a significantly higher mortality than those who are well-nourished.[9] Measurement of REE and RQ has been shown to be helpful in designing nutritional regimens to reduce $\dot{V}_{CO_2}$ in patients with chronic obstructive pulmonary disease (COPD) and patients requiring mechanical ventilation.[1,6,10-15]

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An abstract based on the study reported here was presented by Dr El-Khatib at the OPEN FORUM during the 1995 AARC Annual Meeting in Orlando, Florida.

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Avoidance of excessive nutritional support at the time of weaning from mechanical ventilation can help prevent the development of hypercapnia.[13-15]

There is a growing clinical emphasis on predicting an individual's optimum nutritional requirement. Several studies have been undertaken in the adult population to determine the energy expenditure in critically ill patients.[16-19] However, little is known about the energy expenditure for patients in the pediatric intensive care unit (PICU).

Two methods are commonly used to determine the caloric or energy requirements during mechanical ventilation. The first uses estimated (eg, Harris-Benedict, or HB; World Health Organization, or WHO; or Schofield) equations, which give an estimate of REE as a function of patient's height, weight, age, and gender. The second method involves measurement of resting energy expenditures (MEE) by indirect calorimetry (IC). The results of measurement of REE in mechanically ventilated patients have been shown to be more accurate than published formulas used to predict REE[16,17,20] to reduce the incidence of overfeeding and underfeeding[18,19] and to decrease costs associated with total parenteral nutrition (TPN).[19] Although performing metabolic measurements using an indirect calorimeter is a safe, noninvasive procedure, some hazards or complications might be encountered. Short-term discontinuation of the patient from the ventilator for connection to the IC apparatus may result in hypoxemia, bradycardia, and patient discomfort. Inappropriate calibration or system setup may result in erroneous results leading to inappropriate patient management. In addition to its high costs and the need for highly skilled personnel to perform the IC measurements, variable costs, such as those for desiccants and standard gas mixtures, are associated with many IC systems.

The purpose of this study was to derive new equations for estimation of the REE in children and infants receiving mechanical ventilation in the PICU.

Methods

All patients who received mechanical ventilation in our PICU and who were clinically evaluated with a metabolic cart between 1-1-92 and 3-31-95 were included in the study. Data were obtained from patients' records. The guidelines for performing the metabolic test in our PICU require that the patient weigh ≥ 3.5 kg, be receiving mechanical support with oxygen concentrations (F_{IO_2}) ≤ 0.60, have no audible air leak, not be paralyzed, and be able to achieve a steady state during the test. Steady state is defined as a period in which the variation in respiratory quotient is < 4% for > 5 consecutive minutes. All patients were ventilated with the Servo 900C ventilator (Siemens-Elema AB, Sweden) in the synchronized intermittent ventilatory mode (SIMV). The duration of measurements was at least 20 minutes.

MEE was determined by IC, using a compact, stand-alone, open-circuit, computerized metabolic cart (MetaScope®, Cybermedic, Louisville CO) as shown in Figure 1. The system utilizes a mixing chamber (15 in. × 13.5 in. × 12.5 in.) for exhaled gas collection and includes a drying system to eliminate humidity. Prior to analysis, gases are conducted through a semipermeable membrane system and then routed through desiccant columns to remove the water vapor. The F_{DO_2} is measured before and after each measurement interval. An external oxygen blender is always added to assure delivery of a constant F_{DO_2}. With an indirect calorimeter, energy expenditures are determined by measuring the subject's exhaled gas volume and then analyzing the concentration of oxygen and carbon dioxide in the expired gases.

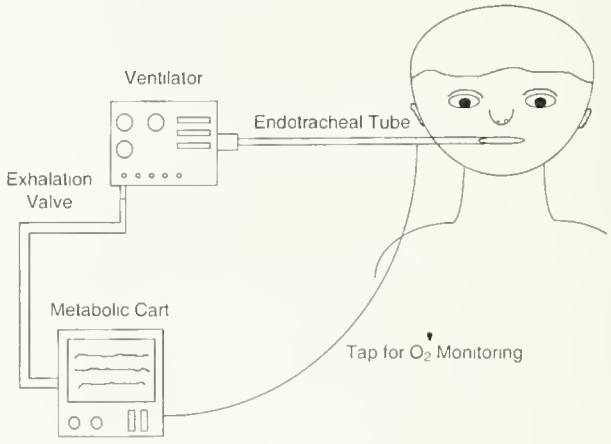

Figure 1. Experimental set-up for indirect calorimetry.

Patients were divided into male and female groups. Multiple linear regression analysis of MEE as function of the patient's height, weight, and age was performed, and statistical significance was set at the 0.05 level. In addition, the HB equation was utilized to estimate the patient's total energy expenditure. Using the Student's *t* test, MEE was compared to the predicted energy expenditure (PEE) by the HB, WHO, and Schofield equations.

Results

Records from 60 patients [34 boys, 26 girls; ranging from 1 month-18 years, with a mean (SD) age of 7 (6.8) years] were included in the study. Fasting, except for intravenous fluid with 5% glucose, was imposed on all patients for 10-12 hours before the test. Some patients were evaluated more than once. Measurements made more than 48 hours apart were considered as separate data points. A total of 77 indirect calorimetry measurements were performed (44 for boys and 33 for girls). Primary underlying diseases were respiratory (23 patients), cardiac (17 patients), neurologic (6 patients), and others (14 patients). As shown in Figure 2, MEE was significantly less than the energy expenditures estimated with either the HB

[mean (SD) = 47.9 (19) vs 60.4 (38) Kcal/kg/day, $p < 0.01$) or the World Health Organization (WHO) equations (47.9 (19) vs 60.5 (33) Kcal/kg/day, $p < 0.01$). MEE was significantly greater than the energy expenditures estimated by the Schofield equations (47.9 (19) vs 41.2 (13) Kcal/kg/day, $p < 0.01$). Multiple linear regression analysis performed on MEE resulted in the following equations:

Boys:

MEE (Kcal/day) = 3.3 Height (cm) + 5.7 Weight (kg) + 75.7 Age (years) + 42.3.
$r^2 = 0.78$

Girls:

MEE (Kcal/day) = 13.9 Height (cm) + 9.5 Weight (kg) − 42.2 Age (years) − 507.
$r^2 = 0.85$

Only moderate agreement was seen between MEE and predicted energy, using the HB, the WHO, and the Schofield equations (Table 1). The highest agreement was achieved with the energy expenditures predicted by our derived equations (Fig. 3).

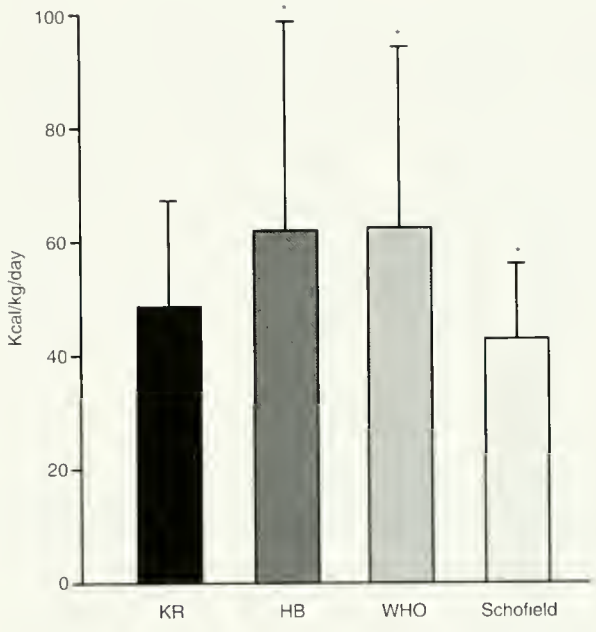

Fig. 2. Measured energy expenditure (MEE) vs the predicted energy expenditures using the Harris-Benedict (HB), the World Health Organization (WHO), the Schofield, and the Khatib-Rosolowski (KR) equations. Values are Mean (SD).* $p < 0.05$ vs MEE ($p < 0.05$ vs KR, paired Student's *t* test).

Discussion

The results demonstrate that in critically ill infants and children receiving mechanical ventilation in the PICU, the HB, WHO, and the Schofield equations were not useful in predicting actual resting energy requirements. There were large variations between predicted and directly measured REE, and MEE was significantly different than PEE.

The HB equation is frequently used to determine energy requirements from weight, height, age, and sex and was originally derived from data obtained from healthy adults in 1919.[21] With the availability of modern computers and advanced mathematical theories, many studies have attempted to improve upon this equation.[16,17,20,22-27] However, most of these studies have been centered on adult patients. Few studies[25-27] have made a serious attempt to evaluate energy requirements in children. Tilden et al[25] measured REE in 18 mechanically ven-

Table 1: Equations for Estimation of Basal Energy Expenditure in Kcal/Day*

	Male Subjects	Female Subjects	
Harris-Benedict	13.752 Wt + 5.003 Ht − 6.755 Age + 66.473	9.563 Wt + 1.850 Ht − 4.676 Age + 655.096	
World Health Organization (WHO)			
3-10 years	22.7 Wt + 495	22.5 Wt + 499	
10-18 years	17.5 Wt + 651	12.2 Wt + 746	
Schofield			
< 3 years	0.167 Wt + 15.174 Ht − 617.6	16.252 Wt + 10.232 Ht − 413.5	
3-10 years	19.59 Wt + 1.303 Ht + 414.9	16.969 Wt + 1.618 Ht + 371.2	
10-18 years	16.25 Wt + 1.372 Ht + 515.5	8.365 Wt + 4.65 Ht + 200	
> 18 years	15.057 Wt + 1.004 Ht + 705.8	13.623 Wt + 2.83 Ht + 98.2	
El Khatib-Rosolowski			
Boys	MEE (Kcal/day) = 3.3 Height (cm) + 5.7 Weight (kg) + 75.7 Age (years) + 42.3		$r^2 = 0.78$
Girls	MEE (Kcal/day) = 13.9 Height (cm) + 9.5 Weight (kg) − 42.2 Age (years) − 507		$r^2 = 0.85$

*Wt: weight in kg, Ht: height in cm, Age: in years.

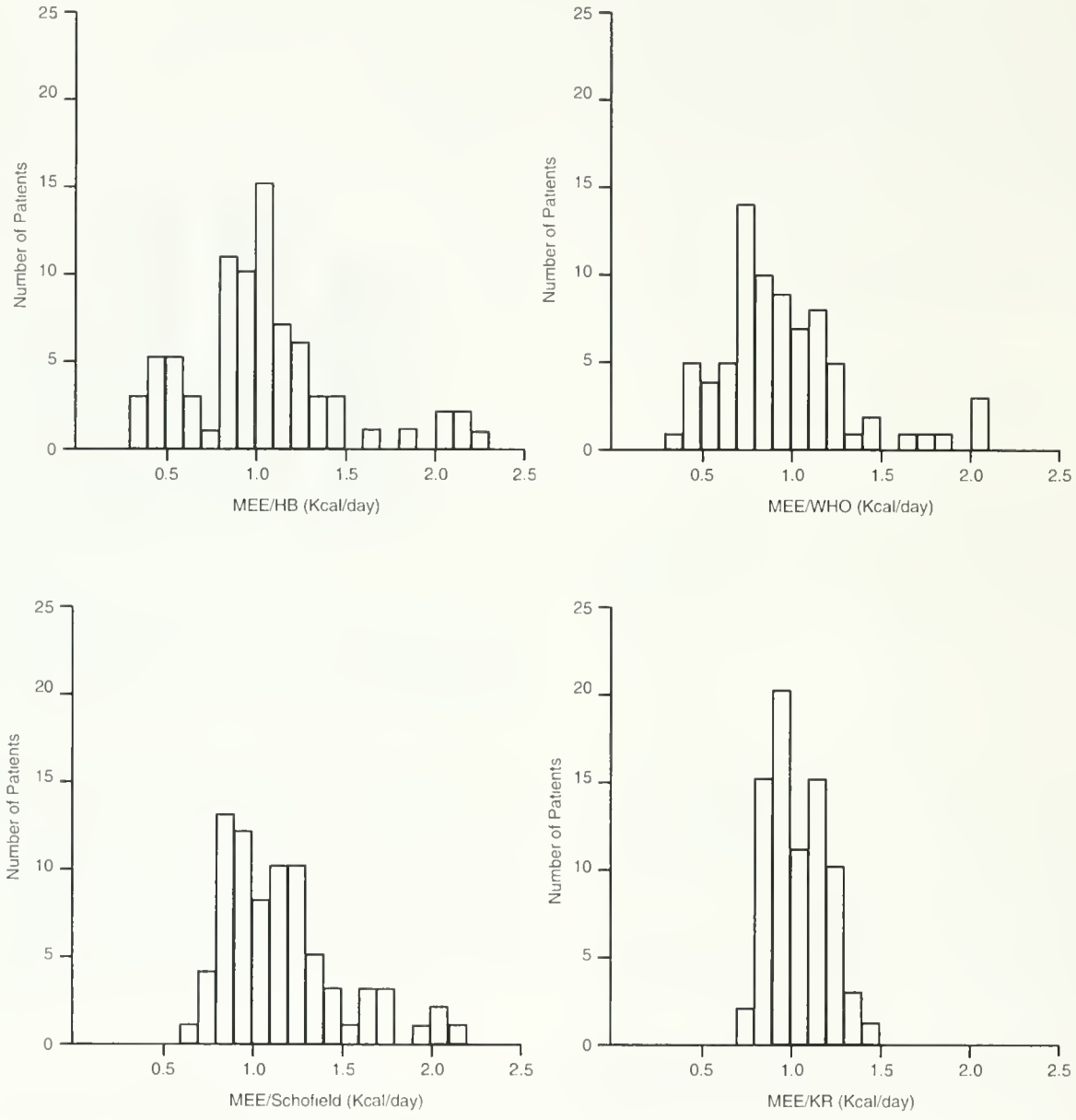

Figure 3. The distribution of the ratio of measured energy expenditure (MEE) to that predicted using the Harris-Benedict (HB), the World Health Organization (WHO), the Schofield, and the Khatib-Rosolowski (KR) equations.

tilated patients aged 2-18 years, using IC. They reported that in the absence of the actual measurement of REE, the recommended energy requirement is 1.5 times basal energy expenditure. In contrast to the current study, Tilden et al did not attempt to derive an equation that could estimate the REE, and they suggested only that the basal energy expenditure formula be shifted upward. Tilden et al also excluded patients who were less than 2 years old. Such patients represent an important portion (32%) of patients included in our study and of patients that we usually see at our institution. Another study by Kaplan et al[26] reported that the HB equation does not provide a good estimation of children who are obese and or who have a nutritional diagnosis of failure to thrive. They suggested that the Schofield and WHO/United Nations University equations could better estimate REE in children. A recent study[27] confirmed the findings of Kaplan et al[26] in healthy children.

In the current study, the comparison of the MEE was only in reference to HB for a number of reasons. The HB equation was the first reported equation and still the one most widely used. It has been shown to be accurate in some patient pop-

ulations—not just for healthy people. Other known equations are for specific age groups.

Multiple linear regression was used to determine the regression equations, and, therefore, they are based on the assumption that a linear relationship exists between REE and the independent parameters (height, weight, and age) that were used to derive the regression. In the current study, this assumption was verified but was not perfectly linear, which is reflected in the r^2 of the final regression.

This study demonstrated that energy expenditures can be measured safely in critically ill pediatric patients. There were no complications associated with the use of the metabolic measurement cart. Air leak around the endotracheal leak was not a problem in this study. Lack of an air leak was confirmed on clinical examination by absence of an audible air leak during mechanical inspiration and during testing by the presence of stable minute-to-minute respiratory quotient values. A recent study[28] showed that in the absence of an audible air leak, energy expenditures can be measured accurately in PICU patients receiving mechanical ventilation.

All patients were studied as a single group, regardless of their diagnostic category. The major disease groups were respiratory, cardiac, and neurologic. However, no patients were comatose and all were hemodynamically stable, receiving mechanical ventilation, and afebrile. Also, these patients had been fasting for 10-12 hours prior to performing the metabolic test. The current study is an initial attempt to derive a universal equation for the prediction of metabolic rate of the critically ill infants and children receiving mechanical ventilation in the intensive care unit. Future attempts could be made to separate this patient population into subgroups based on the disease type and to re-evaluate the current findings.

Due to their high costs and the need for skilled professional staff, IC systems may not be available in most PICUs. Also, even when IC systems are available there are some situations in which metabolic measurements are not accurate. These situations might result from the patient condition or certain bedside procedures and activities, such as peritoneal and hemodialysis,[29] or could result from instability of F_{DO_2} due to changes in source gas pressure and ventilator-blender mixing characteristics.[30] In such cases, the use of the equations derived in the current study might be helpful in estimating REE and lead to more appropriate feeding regimens and mechanical support strategy.

Most of the previously reported equations for energy expenditure estimation were derived with data obtained from adult subjects. Due to the important differences between male and female body habitus and muscle mass, separate equations were derived for male and female groups.[21] Few pediatric studies have looked at boys and girls separately.[26,27] In our study, we reported two different equations for boys and girls. However, we also combined all patients into one group. When this was done, the correlation ($r^2 = 0.74$) was no better than when boys ($r^2 = 0.78$) and girls ($r^2 = 0.85$) were grouped separately. In an attempt to obtain a universal equation, we included all age groups in the multiple linear regression analysis. However, this was preceded by analysis of two age groups (≤ and > than 3 years of age). The obtained results were not significantly different than the current results.

Our study does have limitations. It was a retrospective study encompassing a period of slightly more than 3 years, and the number of subjects and measurements was small, considering the heterogeneity of the group studied. The next logical step is to prospectively validate the derived equations in a similar setting.

In Conclusion

When resting energy expenditure cannot be measured directly, clinicians and other health-care specialists may consider using the equations presented here for infants and children receiving mechanical ventilation in the intensive care unit. These equations may improve the accuracy of an estimated resting energy expenditure and are simple to use, provided that accurate weight, height, and age are available.

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RESPIRATORY CARE

Special Issues

Mechanical Ventilation:
Ventilatory Techniques, Pharmacology,
& Patient Management Strategies
Part 1: April 1996
Part 2: May 1996

Evaluation of a Protective Sheath for Disposable Pulse Oximetry Probes

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Mary Ellen Kern RN MSN, and James M DuBois MHA MIS RRT

BACKGROUND: We evaluated a protective sheath for disposable pulse oximetry probes that is intended by the manufacturer to prolong the life of such probes and to make them reusable from patient to patient. DEVICE: The device is a preformed tape sleeve with an adhesive backing to be placed over a disposable pulse oximetry probe that has been modified by lamination. MATERIALS & METHODS: The study was performed as a prospective clinical trial in a 10-bed multidisciplinary intensive care unit in a 500-bed university hospital. Probe performance was evaluated in 20 adult patients whose oxyhemoglobin saturation was being monitored continuously by pulse oximetry and whose baseline S_{pO_2} was > 90%. All patients had S_{pO_2} measurements recorded by standard disposable pulse oximetry probes and identical probes modified by the protective sheath being evaluated. S_{pO_2} was recorded every 15 minutes for 12 readings/probe/patient, and the readings from the sheathed and non-sheathed probes were compared. New non-sheathed probes were used on each subject, and 5 sheathed probes were alternated among the 20 subjects. RESULTS: Mean S_{pO_2} readings were 97.5% for the non-sheathed probes and 98.1% for the sheathed probes. Differences in mean S_{pO_2} ranged from 0.1% to 2.5% in individual patients. CONCLUSIONS: The protective sheath studied did not interfere with S_{pO_2} measurements in the range tested (> 90%) and allows effective reuse of the disposable pulse oximetry probes studied.
[Respir Care 1996;41(3):197-201]

Introduction

The use of pulse oximetry has become a routine part of monitoring in the intensive care unit (ICU), and disposable pulse oximetry probes have come into wide use for continuous monitoring of oxygen saturations (S_{pO_2}). Although disposable probes may be more comfortable for patients and more convenient for health-care workers than reusable probes, they can add significantly to equipment costs for an ICU because they are not intended for patient-to-patient reuse under ordinary circumstances and because probes may need to be changed (for a variety of reasons) on an individual patient. In this paper, we report on our preliminary experience with a protective sheath for disposable pulse oximetry probes. The primary intent of this sheath is to confer patient-to-patient reusability on oximetry probes that have been regarded previously as disposable. A proposed additional benefit of this sheath is that it may prolong the useful life of probes even for the same patient.

Materials & Methods

Study Design: Prospective clinical trial.

Location: Adult medical-surgical ICU in a 500-bed university hospital.

Patients: 20 patients were studied. All had baseline S_{pO_2} measurements > 90% by oximetry.

Dr Gerber is Director, Intermediate Intensive Care Unit, Assistant Professor of Medicine and Anesthesia, and Director, Critical Care Fellowship Program; Dr Santarelli is Assistant Professor of Medicine; Dr Scott is Associate Professor of Anesthesia; Ms Kern is Clinical Nurse Specialist, Critical Care Medicine and Mr DuBois is Administrative Director, Division of Pulmonary and Critical Care Medicine and Administrative Director, Department of Respiratory Care—University of Medicine and Dentistry of New Jersey/Robert Wood Johnson Medical School at Camden and Cooper Hospital/University Medical Center, Camden, New Jersey.

The authors have no financial interest in the products studied.

Materials: Disposable oximetry probes were utilized to monitor S_{pO_2} in all subjects.* The device tested is a preformed tape sleeve that is placed over a disposable probe that has been laminated with tape. The laminated probe is placed within the sheath, which has an adhesive underside. The sheathed probe is then applied in the routine fashion, using the sheath's adhesive, rather than that of the probe (Fig. 1 A-C). The sheath can be removed from the skin at any time and, if still adhesive, reapplied elsewhere. If the caregiver desires, the probe can be removed from the sheath and reused on the same patient or on another patient.

Methods: Each patient was simultaneously monitored with two disposable pulse oximetry probes on different digits. (Although we used nonadjacent digits whenever practical, Severinghaus and Naifeh[1] have established the absence of interference between pulse oximetry probes even when they are placed on adjacent digits.) One probe was a standard Nellcor D-25 probe, the second a standard probe covered with the sheathing device being tested. Each probe was connected to a separate oximetry module, and the probes were cross-checked against both modules to ensure consistency of readings prior to each trial. The S_{pO_2} reading from each probe was recorded at 15-minute intervals for a total of 12 readings/probe/patient. A new non-sheathed probe was used on each subject, and 5 laminated probes were randomly alternated for use as the sheathed probe (each probe used from 2 to 5 times). Laminated probes were cleaned with a 70% isopropyl alcohol preparation between patients, as suggested by the manufacturer. S_{pO_2} readings obtained with the sheathed probes were compared to readings from the non-sheathed probe, which served as the control. Partial pressure of oxygen as determined from direct analysis of an arterial blood sample was not measured because the purpose of this study was to evaluate the effect of the shield on S_{pO_2} measurements in comparison to measurements made with a standard probe.

Statistical Analysis: Oxygen saturation measurements were assessed using a paired analysis within each individual subject. Agreement between the two devices was evaluated by the method described by Bland and Altman.[2] The upper and lower limits of agreement were calculated by:

$$\text{Upper} = \text{mean difference} + 2\ \text{SD}.$$
$$\text{Lower} = \text{mean difference} - 2\ \text{SD}.$$

Agreement within ±4% was considered adequate because this is the accepted error of measurement with pulse oximetry under ordinary circumstances.[3] Analysis was performed on repeated pairs of measurements within each subject.

The study was approved by the Cooper Hospital/University Medical Center Institutional Review Board as a minimal risk study.

*Suppliers are identified in the Product Sources section at the end of the text.

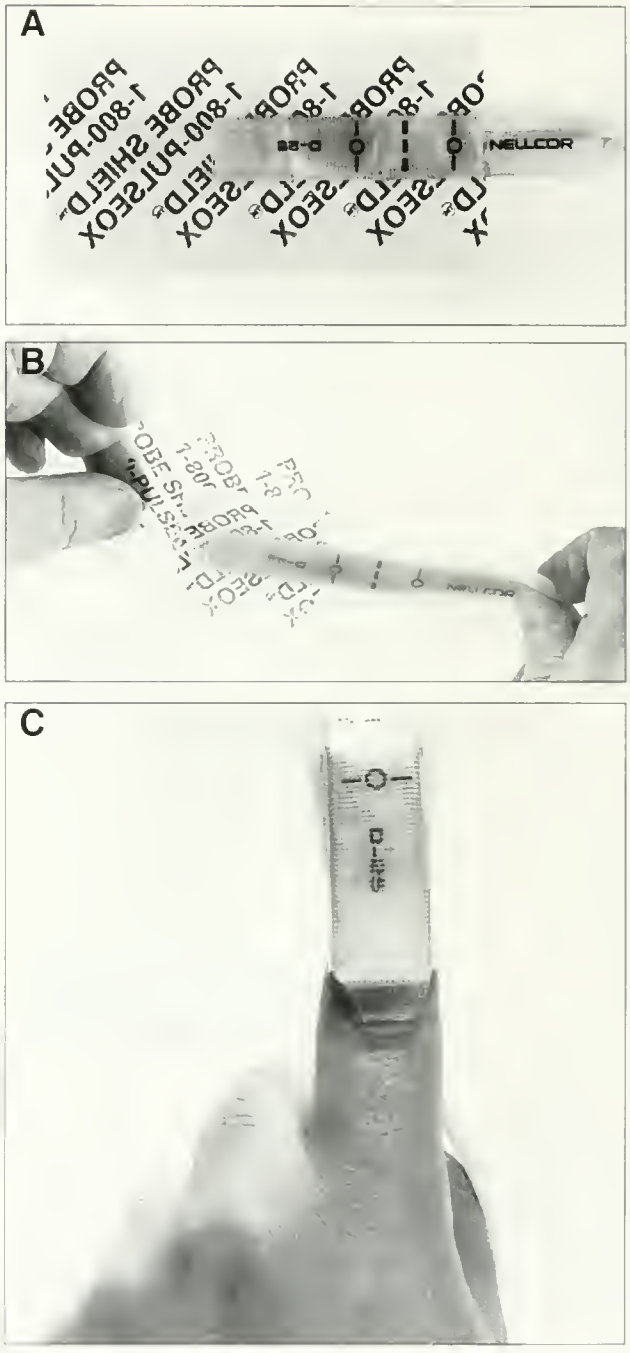

Fig. 1. A. Probe being inserted into sheath (shown partially inserted). B. Sheath with probe fully inserted being removed from backing, exposing adhesive surface of sheath. C. Sheathed probe applied to finger.

Results

Twelve readings from each probe were obtained on each subject, for a total of 480 S_{pO_2} recorded readings, 240 for non-sheathed and 240 for sheathed probes. Differences in mean S_{pO_2} between the sheathed and non-sheathed probes varied

from a low of 0.1% to a high of 2.5%. Overall mean (SD) S_{pO_2} was 97.5% (1.81) for the standard probes and 98.1% (1.61) for the sheathed probes. Readings for each individual probe are presented in Table 1. For the purposes of this study, we considered the readings by the standard, non-sheathed probe as the control. With an accepted error of measurement of ± 4% for pulse oximetry when the arterial saturation is greater than 70%,[3] the measurements of arterial saturation as recorded by the shielded probes must be considered essentially identical with those from the non-sheathed probes. Standard deviations for measurements in individual subjects were similar for the non-sheathed and sheathed probes, indicating that variations in S_{pO_2} as recorded with the sheathed probe were consistent with variations as recorded by the non-sheathed probe. In no case did the differences in saturations as measured by the two probes reach a level that would be considered clinically important (ie, the differences in S_{pO_2} as measured by the two probes would not have resulted in changes in therapy or other clinical interventions).

For 18 of the 20 patients evaluated, agreement between the sheathed and non-sheathed oximetry probes as determined by the Bland and Altman[2] technique was excellent (within ± 4%). For two patients (Numbers 13 and 18), the lower limits of agreement were outside this range, although the upper limits fell within the range. Patient 13 had a lower limit of agreement of −5.77. However, for no paired measurements (between the sheathed and non-sheathed probes) of S_{pO_2} did the difference exceed 4% for this subject, and this was reached only once. The variability of saturation readings from each of the two probes did however result in a large standard deviation. In a similar manner, the difference between the two probes never exceeded 4% for Subject 18, and in this subject the lower limit of agreement was only marginally beyond the predetermined limit, at −4.11. However, as in Subject 13, a large standard deviation resulted from internal variability in saturation readings for the two probes. In this subject in particular, it should be noted, the general trends of S_{pO_2} mirrored each other, going up and down in synchrony.

Table 1. S_{pO_2} Readings for Sheathed and Non-Sheathed Probes

Patient	Probe Type	Probe Number*	Mean (SD) S_{pO_2}
1	Non-Sheathed		98.1 (0.52)
	Sheathed	5	98.7 (0.49)
2	Non-Sheathed		99.3 (1.07)
	Sheathed	3	98.8 (1.75)
3	Non-Sheathed		98.5 (1.00)
	Sheathed	3	99.7 (1.16)
4	Non-Sheathed		100 (0.00)
	Sheathed	3	99.0 (0.85)
5	Non-Sheathed		96.9 (0.90)
	Sheathed	3	97.4 (1.17)
6	Non-Sheathed		99.3 (0.62)
	Sheathed	1	99.8 (0.39)
7	Non-Sheathed		98.4 (0.67)
	Sheathed	1	99.8 (0.34)
8	Non-Sheathed		99.2 (0.94)
	Sheathed	2	98.3 (0.76)
9	Non-Sheathed		95.5 (1.34)
	Sheathed	3	95.6 (1.31)
10	Non-Sheathed		97.6 (0.67)
	Sheathed	4	98.1 (0.67)
11	Non-Sheathed		97.5 (0.52)
	Sheathed	1	97.0 (0.74)
12	Non-Sheathed		95.7 (0.65)
	Sheathed	2	96.8 (0.75)
13	Non-Sheathed		96.4 (1.08)
	Sheathed	1	97.7 (2.57)
14	Non-Sheathed		96.7 (0.65)
	Sheathed	1	96.9 (0.51)
15	Non-Sheathed		97.5 (0.91)
	Sheathed	1	98.5 (1.00)
16	Non-Sheathed		98.7 (0.99)
	Sheathed	2	97.8 (1.12)
17	Non-Sheathed		97.4 (0.52)
	Sheathed	4	99.4 (0.52)
18	Non-Sheathed		95.3 (1.07)
	Sheathed	4	97.8 (0.62)
19	Non-Sheathed		93.5 (1.13)
	Sheathed	4	95.3 (1.19)
20	Non-Sheathed		98.8 (0.58)
	Sheathed	5	98.6 (0.52)

* Refers only to shielded probes, Numbers 1-5.

Discussion

Pulse oximetry has become an extremely common monitoring technique in the ICU as well as in other acute patient care areas, such as the operating room and the emergency department. Such monitoring is included in recent guidelines established by the Society of Critical Care Medicine with regard to patients receiving continuous oxygen therapy[4] or those in respiratory failure requiring mechanical ventilation[5] and has been addressed in Clinical Practice Guidelines published by the American Association for Respiratory Care.[6-9] Because of the general acceptance of pulse oximetry as a reliable and useful method of monitoring the oxygenation status of patients, the use of disposable pulse oximetry probes has become a high-dollar financial obligation for hospitals.

Efforts to decrease the costs of disposable pulse oximetry probes for institutions have been undertaken. Published studies have explored the feasibility as well as the constraints of reusing nominally disposable oximetry probes.[10] The question of reusability revolves around two distinct issues, namely the infectious risk associated with reusing probes between patients and the ability of these probes to continue to function normally for extended periods of time and after being applied, removed, and reapplied to patient after patient. Data suggest that routine cleaning procedures may not be adequately effective in eliminating contamination of previously used probes by pathogenic organisms,[11,12] although the association of such contamination with the spread of nosocomial infection has

not been well documented.[13] Reuse of disposable probes has been shown to be clinically feasible,[10] although technical complications (such as obscuration of the optical windows on the probes by disrupted adhesive) may limit such reuse.[14]

The device we studied attempts to resolve both of these issues. Because the sheath covers the probe and prevents direct contact with the patient, questions of probe contamination and transmission of infectious agents from patient to patient are minimized. Nevertheless, it is recommended by the manufacturer that each probe be cleaned with 70% isopropyl alcohol between patients. The issue of reusability is addressed by the product as well. By enclosing the oximetry probe in a sheath, it is protected from damage by factors such as perspiration and exogenous substances. Also, because it is the removable sheath that is taped to the skin rather than the probe directly, loss or other derangement of the adhesive part of the probe is not an issue. Should the probe have to be relocated to another site on the patient, or should the sheath have to be changed on the same patient, the projected $3 cost of the sheath is far less than the $10-15 cost of a new oximetry probe. In this study we reused the laminated, sheathed probes from two to five times each without adversely affecting their ability to accurately detect S_{pO_2}, demonstrating the utility of this device for that purpose.

The potential for cost savings with a device of this type is fairly obvious. One report relates institutional costs of $168,000 annually for 12,500 disposable oximetry probes.[10] In another study, a change from using only disposable probes to using only reusable probes in an operating room yielded savings of $8,232 in 1 month, and projected annual savings of $66,600.[15] Based on these findings it can be seen that even a modest reduction in the total number of probes used can result in substantial savings. In addition, current practices for reusing nominally disposable oximetry probes call for reprocessing, which includes autoclaving and repackaging. However, this practice should not be necessary with the product tested because it is the sheath rather than the probe that is the patient-specific component.

Although we are aware of equipment costs and the number of procedures in our own unit, we have only projected potential savings and, thus, are not able to report actual cost savings to our institution. This is a limitation of our study and the subject of ongoing analysis. Likewise confining the study to patients with $S_{pO_2} > 90\%$ is also a limitation. The effect of the sheath on accuracy at lower saturations still needs to be established, although preliminary data suggest that it does not adversely affect the accuracy of the S_{pO_2} readings, even at extremely low levels.[16]

We believe that the data indicate that the Medical Taping Systems Probe Shield protective sheath for disposable pulse oximetry probes does not interfere with the accuracy of measurements of S_{pO_2} within the normal (> 90%) range, as in the population studied. Given this apparent clinical reliability and the potential for cost savings with its use, we suggest that strong consideration be given to adopting the use of this product in an effort to reduce the costs of pulse oximetry without adversely affecting patient care.

ACKNOWLEDGMENTS

We thank Medical Taping Systems for providing the laminated oximetry probes and all Probe Shields and SpaceLabs Medical for providing the 90489 oximetry modules and the 90308 PC Express portable monitor for use in this project.

PRODUCT SOURCES

Protective sheath:
Probe Shield Medical Taping Systems, Glenmoore PA

Laminating tape:
Medical Taping Systems, Glenmoore PA

Disposable pulse oximetry probe:
Nellcor D-25, Nellcor, Hayward CA

Pulse oximetry module:
SpaceLabs 90489, SpaceLabs, Redmond WA

Portable oximetry module:
PC Express 90308, SpaceLabs, Redmond WA

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CORRECTION

Figure 4 was omitted from Del Bufalo C, Morelli A, Bassein L, Fasano L, Quarta CC, Pacilli AMG, Gunella G. Severity scores in respiratory intensive care: APACHE II predicted mortality better than SAPS II. Respir Care 1995;40(10):1042-1047. It is printed here in its entirety. We regret the error.

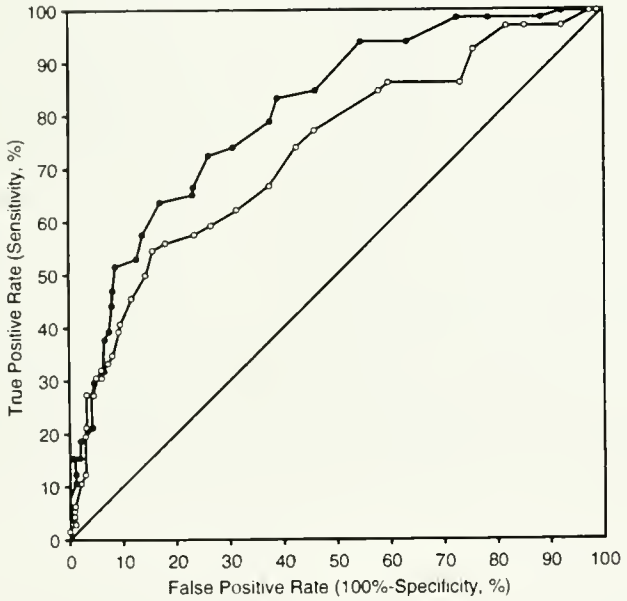

Fig. 4. Receiver Operating Characteristic (ROC) curves for APACHE II and SAPS II. The APACHE II ROC (—●—) area is 80.88%, with a standard error (SE) of 2.89%. The SAPS II ROC (—○—) area is 73.52%, with an SE of 3.61%. The area under the ROC curve for the APACHE II system was significantly greater ($p < 0.01$) in our intensive care unit patients with respiratory disease.

Corrected reprints are available from the author: Cimino Del Bufalo MD, Servizio di Fisiopatologia Respiratoria, Unità di Terapia Intensiva Respiratoria, Ospedale Policlinico S. Orsola, Via Massarenti 9, 40138 Bologna, Italy.

A National Survey on the Prevalence of Cigarette Smoking in Pediatric Respiratory Care Departments and Its Effect on Smoking Cessation Efforts

Teri Nikolai Wilson RCP RPFT RRT and Robert C Cohn MD

BACKGROUND: The effects of second-hand smoke on children is a major health concern. A number of studies have shown impaired lung function and higher rates of respiratory tract illnesses in children exposed to passive cigarette smoke. Pediatric respiratory care practitioners (RCPs) are often the first professionals to evaluate and treat these patients. It is not clear how many pediatric RCPs are smokers or how their approach to patients and family members who smoke is influenced by their smoking status. METHODS: We surveyed 59 respiratory care department managers of children's hospitals and other pediatric facilities. RESULTS: The response rate was 56%. The mean number of RCPs/department was 49. On average, the departments reported that 10% of RCPs were smokers. Seventy percent of departments do not routinely ask patients and their family members about smoking habits, 67% of RCPs do not routinely counsel parents about the negative consequences of second-hand cigarette smoke, and 94% do not have written information for patients and families about passive cigarette-smoke exposure. Only 6 (18%) reported the availability of a cigarette smoking-cessation program. In departments where ≥ 20% of the staff smoke (n = 3), no routine effort to educate parents by RCPs was reported. In departments reporting < 20% smokers (n = 30), 13 report routine efforts to educate parents about the health effects of passive smoke exposure. CONCLUSION: We conclude that RCPs need to become more active in their efforts to improve patients' health by offering information and/or programs to promote smoking cessation and by becoming nonsmoking role models. [Respir Care 1996;41(3):202-205]

Background

The effects of passive cigarette-smoke exposure on nonsmokers has gained national attention. The Environmental Protection Agency has classified cigarette smoke as a class A carcinogen, a distinction shared with only 15 other substances including radon, asbestos, and benzene.[1] The effects of second-hand smoke on children have been well documented and have been reported to cause impaired pulmonary function, increased airway hyperreactivity, and higher rates of lower respiratory tract infections.[2-4] Approximately 70% of children in the United States live in homes with at least one active cigarette smoker.[5] Cigarette smoke generated by parents who smoke at home can aggravate symptoms in children with asthma and can even trigger asthma attacks.[6]

Many authors recommend smoking cessation for parents.[4-9] We were surprised to find few data on the prevalence of cigarette smoking among respiratory care practitioners (RCPs)

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in the U.S. and, especially, among those who care for children.[10-12] To determine the prevalence of cigarette smoking among pediatric RCPs and to determine whether smoking-cessation counseling is provided by pediatric respiratory care departments with a large percentage of smokers, we surveyed the managers of respiratory care departments in children's hospitals and other pediatric facilities.

Methods

Using the registration list of the National Association for Children's Hospitals and Related Institutions (NACHRI), a list of 147 children's institutions was obtained.[13] Fifty-nine were chosen to receive the survey based on the existence of an active respiratory care department. Figure 1 lists the survey questions. The survey was specifically sent to the respiratory care department manager to obtain their perception of the smoking status and counseling habits of the RCPs.

The surveys were mailed with an accompanying letter requesting that the department manager respond to the survey. Surveys were returned by facsimile. The data were tallied by one of the investigators (TNW) and recorded.

The data were broken into 2 descriptive groups: departments with ≥ 20% RCP smokers and departments with < 20% RCP smokers. The prevalence of smoking and provision of smoking-cessation counseling among RCPs was drawn from this information. Data were not subjected to statistical analysis because of the small sample.

Results

Thirty-three responses (56%) were received from hospitals in 22 different states. Five responses were received from Western States, 3 from Southwestern States, 4 from North-Central States, 5 from Mid-Atlantic States, 3 from South-Central States, 3 from Southeastern States and 10 from Northeastern States. Of the 33 institutions that responded, 32 are not for profit, and 30 are short-term general (average length of stay < 30 days) children's hospitals. Average annual admissions were 9,075 (range 40-16,909), with an average bed size of 199 (range 27-297). Table 1 lists these characteristics for responders and nonresponders.

Eighty-five percent of departments provide a smoking area for employees, 88% provide a smoking area for families, and one respondent has a smoking area that is used by both staff and families. The mean number of RCPs/department was 49 (range 4-90). On average, responding departments report that 10% of RCPs are smokers (range 0%-34%).Of respondents, 70% reported that RCPs do not routinely ask family members about their smoking habits, and 67% responded that RCPs do not routinely counsel parents about the negative consequences of second-hand smoke. The majority (94%) of respiratory care departments do not provide written information about the effects of second-hand smoke to family members. Only 6 hospitals (18%) reported the availability of a smoking-cessation program—3 of these are available only for employees. Smoking-cessation programs are being developed in 2 departments.

Table 1. Characteristics of Survey Responders and Nonresponders

	Responders	Non Responders
Ownership		
Not-for-profit	32	25
Church-operated	0	1
Partnership	1	0
Scope of Service		
Children's general	30	16
Children's rehabilitation	2	2
Children's other specialty	1	0
Children's orthopedic	0	1
Children's unit of general	0	2
Length of Stay*		
Short term	30	22
Long term	3	4
Average Beds	199 (range 27-297)	144 (range 7-198)
Average Admissions/Year	9,076 (range 40-16,909)	5,157 (range 19-11,456)

*Short term = < 30 days, long term = ≥ 30 days

The responses were summarized in 2 groups: respiratory care departments with ≥ 20% RCP smokers and those with < 20% RCP smokers. In departments with ≥ 20% RCP smokers (n = 3), no (0%) routine effort by therapists to counsel parents was reported. In departments reporting < 20% RCP smokers (n = 30), 13 (39%) reported routine efforts to educate parents about the negative consequences of second hand smoke.

Discussion

The morbidity among pediatric patients from passive smoke exposure has been extensively reviewed showing substantial health effects on the respiratory system, cardiovascular system, fetus, and newborn.[4] It is interesting to note that department managers responding to this survey reported a 10% smoking prevalence among RCPs, considering that others report this prevalence to be close to that of the general public —about 26%.[10-12] This discrepancy could be due to underestimation of smoking prevalence by the department managers, or it could be that there is a different smoking prevalence among RCPs who care for adults.

Primary prevention—ie, preventing recruitment of new smokers—is the most effective strategy to decrease smoking prevalence. Secondary prevention—ie, smoking cessation for current smokers—must be emphasized because cul-

Survey of Respiratory Therapy Departments Regarding Smoking Cessation Efforts

1. Is there a smoking section on hospital property for employees? (Yes) (No)
2. Is there a smoking section on hospital property for visitors? (Yes) (No)
3. How many respiratory therapists are employed in your department? _____
4. What percentage of respiratory therapists employed at your hospital currently smoke? _____
5. Do respiratory therapists employed by the hospital routinely ask family members about their smoking habits? (Yes) (No)
6. Do respiratory therapists routinely counsel family members regarding the health effects of second-hand smoke? (Yes) (No)
7. Do respiratory therapists routinely provide written information regarding the health effects of second-hand smoke? (Yes) (No)
8. In what setting does this education take place?
   Emergency room _____, Outpatient setting _____, Inpatient setting _____.
9. Does your hospital offer any organized classes for smoking cessation? (Yes) (No)
10. What is the fee for the program? _____
11. How many sessions are included in the program? _____

Your Name _____

Hospital _____

Address _____

_____

Phone Number _____

Is there a specific person I can contact for further questions regarding smoking cessation programs?

_____

Figure 1. Survey distributed to 59 children's institutions with active pediatric respiratory care departments.

dren whose parents smoke are also likely to become smokers in the future.[4] An aggressive cigarette smoking-cessation public health program directed at both parents and children is needed. This requires the strong support of respiratory care practitioners, who must first become role models and advocates by exhibiting smoke-free lifestyles. A survey by Robbins, Albright and Deutsch[11] explored the attitudes of RCPs towards their role in smoking cessation. They found that more nonsmokers and former smokers agreed that nonsmoking advocacy should be part of their job responsibilities than did current smokers.

The credibility of an RCP smoker in the role of a stop-smoking advocate is questionable. Tobacco smoke contamination of clothing, hair, and skin may linger and be intense.[14] Long after a person has left a smoke-filled room, the odor of cigarettes on hair and in clothing fabric may persist. Smokers usually are not sensitive to these odors, and this can be an important problem—one that is largely overlooked by RCPs, often the first health professionals to assess and treat children with acute and chronic respiratory problems.[6]

Survey data may help document patterns of practice. This survey documents a lack of active smoking-cessation efforts by pediatric respiratory care departments, who are dealing not with the primary smoker but the child who is an involuntary smoker. We recognize that our asking the department manager questions about the behavior of RCPs may not give a totally accurate picture, but does raise the issues that surround RCP behavior related to smoking cessation.

As more pediatric respiratory care departments become involved in smoking-cessation efforts, more detailed documentation of the habits of RCPs in pediatric institutions and their level of smoking-cessation advocacy is warranted. We hope our study provides the impetus for respiratory care departments at children's hospitals and pediatric facilities to assess the smoking habits of staff members and develop smoking-cessation and -prevention programs.

We conclude that RCPs need to become more active in their efforts to improve patient's health by offering information and/or programs to promote smoking cessation. The importance of positive role models cannot be overstated. RCPs who smoke must first examine the message they send to the public, stop smoking, and get involved in the efforts to promote smoking cessation.

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From the Centers for Disease Control:

Leads from the Morbidity and Mortality Weekly Report
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Case-Control Study of HIV Seroconversion in Health-Care Workers After Percutaneous Exposure to HIV-Infected Blood—France, United Kingdom, and United States, January 1988-August 1994

MMWR 1995;44:929-933; one table omitted

Health-care workers (HCWs) are potentially at risk for human immunodeficiency virus (HIV) infection through occupational exposures to blood. Although prospective studies indicate that the estimated risk for HIV infection after a percutaneous exposure to HIV-infected blood is approximately 0.3%,[1,2] factors that influence this risk have not been determined. To assess potential risk factors, CDC, in collaboration with French and British public health authorities, conducted a retrospective case-control study using data reported to national surveillance systems in the United States, France, and the United Kingdom. This report* describes the study and summarizes results that suggest that risk factors for HIV transmission include certain characteristics of the exposure and the source patient; in addition, postexposure use of zidovudine (ZDV) by HCWs was associated with a lower risk for HIV transmission.

Case-HCWs had a documented occupational percutaneous exposure to HIV-infected blood (i.e., a needlestick or a cut with a sharp object [e.g., scalpel or lancet]), HIV seroconversion temporally associated with the exposure, and no other concurrent exposure to HIV. Control-HCWs had a documented occupational percutaneous exposure to HIV-infected blood and were HIV seronegative at the time of exposure and at least 6 months later. Case-HCWs were identified through reports to national surveillance systems for occupationally acquired HIV infection operated by CDC, in cooperation with state and local health departments (United States), the National Public Health Network (Reseau National de Sante Publique) (France), and the Public Health Laboratory Service Communicable Disease Surveillance Center (United Kingdom). Control-HCWs were identified through reports to a passive surveillance project maintained by CDC since 1983 that includes data from approximately 300 health-care institutions in the United States.[1]

The study included all case-HCWs reported in the United States whose exposure occurred during January 1988-August 1994 and all control-HCWs exposed after January 1988 whose 6-month follow-up evaluation was completed as of August 1994. Case- and control-HCWs reported in the United States before 1988 were excluded from the analysis because information on some variables was not routinely collected and because postexposure use of ZDV was infrequent before 1988.[1] For similar reasons, analysis was limited to case-HCWs reported in France since 1990 and in the United Kingdom since 1989.

Information obtained about HCWs included age; sex; occupation; work location; and whether postexposure antiretroviral agents were offered, whether they were used, how long after the exposure the first dose was used, daily dosage, and duration of treatment. Information about source patients included stage of HIV infection (acquired immunodeficiency syndrome [AIDS], symptomatic, or asymptomatic), use of antiretroviral drugs at the time of the HCW's exposure, and presence of terminal illness (i.e., death because of AIDS within 2 months after the exposure).

Information about exposures included the type of device involved, gauge of hollow-bore needle, type of procedure being performed, whether the procedure was an emergency, use of gloves, time from use of the device to exposure, presence of visible blood from the source patient on the device, and severity of

*Reported by: State and territorial health depts. CDC Cooperative Needlestick Surveillance Group. D Abiteboul, MD, Institut National de Recherche et de Securite and Groupe d'Etude sur le Risque d'Exposition au Sang, Paris, F Lot, MD, Reseau National de Sante Publique, Saint Maurice, France. J Heptonstall, MRCPath, Public Health Laboratory Service Communicable Disease Surveillance Center, London, United Kingdom. Div of HIV/AIDS Prevention, National Center for Prevention Svcs; Hospital Infections Program, National Center for Infectious Diseases, CDC.

injury. Severity of injury was defined as superficial (surface scratch, no blood appeared), moderate (penetrated skin and blood appeared), or deep (deep puncture or wound with or without bleeding).

The study included 31 case-HCWs (23 from the United States, five from France, and three from the United Kingdom) and 679 control-HCWs (who were from 190 of the reporting health-care institutions). Of the 31 exposures sustained by case-HCWs, 29 (94%) were needlesticks (all with hollow needles) and two (7%) involved other sharp objects. Of the 679 exposures sustained by control-HCWs, 620 (91%) were needlesticks (including 594 hollow and 26 solid needles) and 59 (9%) involved other sharp objects.

For both case- and control-HCWs, 74% were exposed during 1990-1994, when ZDV postexposure use had become more common.[1] During 1990-1994, 17 (81%) of 21 case-HCWs had been offered ZDV, and from September 1990 (when collection of information on whether ZDV was offered to control-HCWs became routine) through 1994, 268 (79%) of 338 control-HCWs were offered ZDV. ZDV postexposure prophylaxis was used by nine (29%) case-HCWs and 247 (36%) control-HCWs (crude odds ratio = 0.7; 95% confidence interval = 0.3-1.7). Regimens for case- and control-HCWs generally were 1,000 mg/day for 3-4 weeks; the small number of case-HCWs who used ZDV precluded assessment of differences in ZDV regimens between case- and control-HCWs.

All variables that were statistically significant in the univariate analysis and variables potentially important for prevention (e.g., use of gloves, whether ZDV was offered, and whether ZDV was used) were examined using logistic regression analysis. Based on this analysis, factors associated with HIV transmission included a deep injury, device visibly contaminated with the source patient's blood, procedures involving a needle placed directly in a vein or artery, and terminal illness in the source patient. In addition, case-HCWs were significantly less likely to use ZDV than control-HCWs (adjusted odds ratio = 0.2, $p < 0.01$).[†] The crude odds ratio for ZDV use differed from the adjusted odds ratio because ZDV use was more frequent, among both case- and control-HCWs, for exposures characterized by the other factors. All factors in the model also were significant when the analysis was restricted to case-HCWs from the United States.

The degree of susceptibility to ZDV of HIV strains from source patients and case-HCWs is unknown. Information about antiretroviral treatment for source patients was available for seven case-HCWs and 124 control-HCWs who had used ZDV; five (71%) case-HCWs and 87 (70%) control-HCWs were exposed to blood from source patients who had been receiving ZDV at the time of the exposure.

CDC Editorial Note: The findings in this report indicate that, among the HCWs in this study, an increased risk for HIV infection following percutaneous exposures to HIV-infected blood was associated with three factors. First, the risk increased if the exposure involved a larger quantity of blood, indicated by (1) a device visibly contaminated with the patient's blood, (2) a procedure that involved a needle placed directly in a vein or artery, or (3) a deep injury. Second, the risk increased for exposures to blood from source patients with terminal illness, probably reflecting the higher titer of HIV in blood late in the course of AIDS or other factors, such as the presence of syncytia-inducing strains of HIV.[3,4] Finally, the analysis of these data suggested that use of ZDV postexposure may be protective for HCWs. After controlling for other factors associated with HIV transmission risk, the model indicates that the risk for HIV infection among HCWs who used ZDV was reduced by approximately 79% (95% CI = 43%-94%) (based on adjusted odds ratio = 0.21; 95% CI = 0.06-0.57). However, the limitations of the study design must be considered when interpreting these results.

A retrospective case-control study is not the optimal study design for assessing ZDV efficacy. The optimal approach—a prospective, placebo-controlled trial—has not been possible because of the requirement for a large number of HCWs and the relatively low rate of HIV seroconversion following occupational exposure.[1] The findings of this study also are subject to at least five potential limitations.

First, case- and control-HCWs were identified using different data sources. Second, if control-HCWs were more likely to have been offered or encouraged to use ZDV, then use of the drug might be statistically associated with lack of HIV seroconversion, even if ZDV is not truly protective; however, available evidence does not suggest that control-HCWs were more likely than case-HCWs to have been offered ZDV. Third, reporting bias may have resulted if HCWs preferentially reported exposures that they believed were more likely to result in HIV transmission; this tendency presumably would be similar for case-HCWs and control-HCWs. Fourth, ascertainment bias may have affected some data, particularly subjective variables such as severity of injury, because information for control-HCWs was obtained prospectively soon after exposure but information for most case-HCWs was obtained after HIV seroconversion; however, for most variables evaluated,

[†] Information on terminal illness in the source patient was missing for 19% of case-HCWs and 48% of control-HCWs; information on visible blood on device was missing for 3% of case-HCWs and 6% of control-HCWs. By recoding the missing values to zero and including missing value indicator variables for these factors in the model, these HCWs were retained in the analysis and their potential confounding influence could be assessed. No significant interactions were found among the risk factors in the model or between the risk factors and the missing value indicators. When all HCWs with missing values for any of the factors were excluded from the analysis, all of the factors remained significant, with similar adjusted odds ratios but larger confidence intervals.

objective documentation from incident reports and medical records was available. Finally, number of case-HCWs evaluated was small.

Although failures of postexposure ZDV to prevent HIV infection in HCWs have been documented,[1] this is the first study of HCWs exposed to HIV that assesses the effectiveness of ZDV as postexposure prophylaxis. Studies involving animals have yielded inconclusive results.[5] In studies involving humans, ZDV was reported to reduce the rate of perinatal HIV transmission[6] and to be beneficial in treating early HIV infection[7]; however, the implications of these results for postexposure prophylaxis are uncertain. The short-term toxicity of ZDV in HCWs primarily has been gastrointestinal discomfort and fatigue.[1,2,5,8]

ZDV is not approved by the Food and Drug Administration for use as postexposure prophylaxis. In a previous statement, the Public Health Service (PHS) concluded that a recommendation could not be made for or against the use of ZDV postexposure prophylaxis because of limited knowledge regarding its efficacy and toxicity.[9] PHS recommends that HCWs who may be at risk for occupational exposure to HIV infection be informed of the considerations pertaining to the use of ZDV for postexposure prophylaxis, including the risk for HIV transmission after the exposure, factors that may increase or decrease this risk, and the limited knowledge regarding the potential efficacy and toxicity of ZDV postexposure prophylaxis.[9] If a decision is made to use postexposure prophylaxis, it should be initiated promptly.[9] PHS is evaluating the implications of the study summarized in this report and other available information in assessing the possible need for revision of recommendations for managing occupational exposure to HIV—particularly regarding postexposure use of antiretroviral agents.

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Persistent Cough in a Once-Wounded War Veteran

Ali Emad MD

Case Summary

A 28-year-old man, who had sustained a bullet wound in the war in 1986, now complained of a severe paroxysmal cough. At the time of his injury, he had undergone exploratory abdominal surgery, which showed a perforated right diaphragm and lacerated liver; both were repaired, and the patient was relatively well up to 40 days after surgery. Thereafter, he gradually developed dyspnea on exertion. Chest radiographs revealed the elevation of the right hemidiaphragm and a right-side pleural effusion. He underwent another abdominal operation during which the liver was found to be herniated through the diaphragm; the liver was reduced into the abdomen, and the diaphragm was repaired. Twenty days after the second abdominal operation, the patient developed a cough that persisted until 1994. This patient was referred to our institution because the cough had worsened and become paroxysmal. Elevation of the right hemidiaphragm had been detected on several chest radiographs and was believed due to sluggish movement of the diaphragm.

The only abnormality on the physical examination was decreased breath sounds in the right-lung base. Elevation of the right hemidiaphragm on the chest radiograph was again noted. Severe narrowing of the right-middle-lobe bronchus and mild narrowing of the right-lower-lobe bronchus was detected during bronchoscopy. Selective right-lung bronchography was performed (Fig. 1).

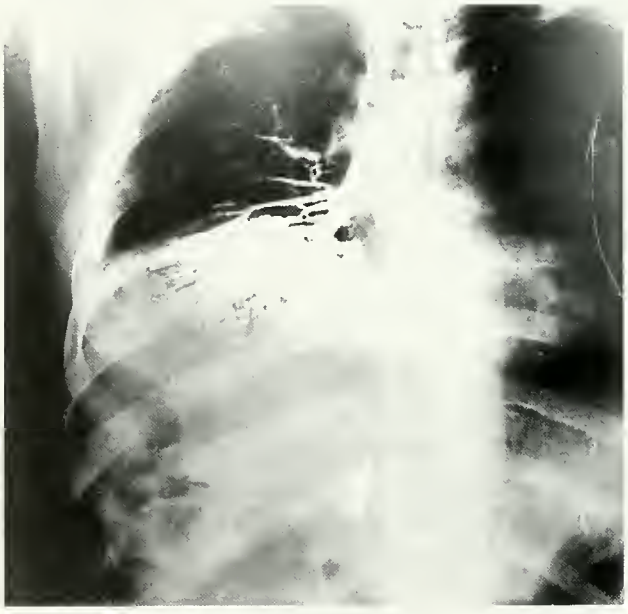

Fig. 1. Bronchograph of a 28-year-old man with chronic, persistent, and paroxysmal cough.

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How would you answer these questions?

What are the most striking features of this bronchogram? _____

_____

What diagnoses should be considered? _____

_____

When is bronchography indicated? _____

_____

Answers and discussion on next page

Striking Features of the Bronchogram

The most striking feature of this bronchogram is the marked elevation of the right hemidiaphragm. In healthy subjects, the right hemidiaphragm is about 1-3 cm higher than the left.[1] The main and lobar bronchi appear narrowed.

In addition, an opaque object—a bullet— can be seen in situ in the medial aspect of the right-lung base.

The Diagnoses To Be Considered

Unilateral elevation of a hemidiaphragm may be seen in a number of conditions. Most of them are intrathoracic conditions and lead to loss of lung volume—atelectasis and hypoplastic lung—and require lobectomy or pneumonectomy. Subphrenic abscess,[2] distended abdominal viscera,[3] and abdominal masses may also cause elevation of the hemidiaphragm. Diaphragmatic hernias through the foramen of Morgagni or Bochdalek may produce symptoms similar to those associated with elevation of a hemidiaphragm.[4]

One of the major causes of unilateral elevation of a hemidiaphragm is a subpulmonic effusion.[5] This diagnosis is usually established with a lateral-decubitus chest radiograph that shows a gravity-dependent fluid level.

Phrenic nerve palsy, which may be idiopathic or caused by primary lung tumors, mediastinal masses or surgery, irradiation, cervical disk herniation,or herpes zoster, are some of the common causes of elevation of a hemidiaphragm.[6] Pulmonary embolism,[7] eventration of the diaphragm,[8] traumatic rupture of the diaphragm,[9] diaphragmatic tumor,[10] and lung hepatization are other less common causes of unilateral elevation of a hemidiaphragm. Phrenic nerve palsy can be diagnosed using fluoroscopy, which can reveal the paradoxical motion of the diaphragm with sniffing and coughing, especially when the patient is in the side-lying position.[11]

Indications for Bronchography

Bronchography seems to be useful for evaluating morphologic changes in the bronchial tree in certain patients, such as those with bronchiectasis. In fact, the main application for bronchography is for diagnosis of bronchiectasis.[12] Despite recent advances in diagnosis of bronchiectasis by means of computerized tomography (CT) of the chest,[13,14] bronchography is still the gold standard test. This is especially true for diagnosis of central bronchiectasis in patients with allergic bronchopulmonary aspergillosis.[15] Bronchography can be useful for diagnosis of congenital tracheal stenosis,[16] Swyer-James syndrome,[17] bronchial disruption due to blunt chest trauma,[18] diverticulosis of the main bronchi,[19] agenesis of the right upper lobe,[20] and for demonstration of successful dilation of bronchial stenosis by implantation of endoprostheses (stents).[21] Bronchography should be performed when bronchoscopy and CT of the chest fail to show radiographically occult carcinomas.[22]

Bronchography can be performed by instillation of a contrast media via the transtracheal route or cricothyroid puncture into the tracheobronchial tree under fluoroscopic control.[23,24] For an optimal visualization of the segmental and subsegmental airways, anesthesia of the endobronchial tree must be adequate, otherwise the patient's cough may make it difficult to do a proper bronchography. Though unpleasant for the patient, a selective or total bronchography via a fiberoptic bronchoscope is safe, simple, quick, and well-tolerated.[25,26]

The contrast medium commonly used is propyliodone (Dionosil). The usual volume required is 10-15 mL. However, its use is not without hazards because it may induce allergic reaction ranging from bronchospasm to death. Other reported complications are pulmonary collapse[27] and a temporary impairment of ventilation and diffusion.[28] Whereas bronchography was commonly used in the past, nowadays, many clinicians avoid it.[29]

In this patient, we suspected a process of persistent hepatization of the right-lower lobe. Thoracotomy was performed. The right lower lobe was found to be attached to the diaphragm. Both the right-middle and right-lower lobes were resected. Histopathologic findings showed a red hepatization of the resected lung tissue (Fig. 2) in our patient, which may be attributed to his trauma and repeated infections. His hospital course (8 days) was uneventful. Now, 8 months after lung resection, the patient is doing well and has no complaint.

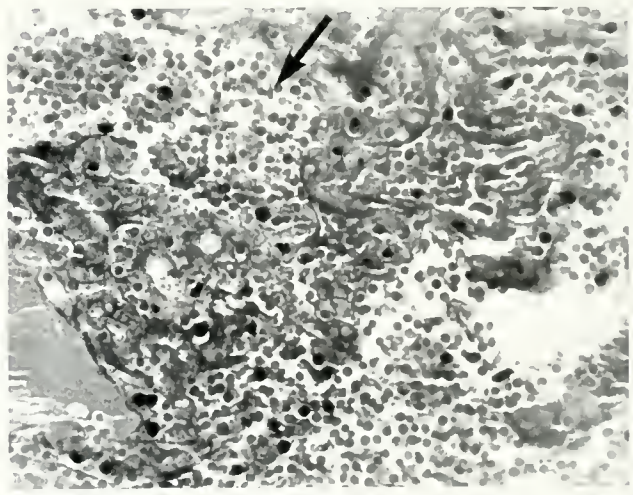

Fig. 2. A magnification (approximately 200X) of resected lung tissue showing red hepatization—sites of recent invasion undergoing active inflammation (hemorrhage), usually peripheral areas of the lesion and gray hepatization—sites near the center or origin of the lesion made a gray color by consolidated leukocytes and lung tissue. The arrow points to the red blood cells, which have filled up all the alveoli.

Lung Hepatization

Hepatization means a transformation of a lesion into a liver-like mass. In the lung, a part of the parenchyma may be solidified as a liver-like mass during a process of pneumonitis.[30,31] The pneumonitis eventually subsides but the lung injury is not reversible. The transformed (hepatisized) lung tissue cannot participate in gas exchange.[31,32]

Persistent hepatization of lung tissue is rare. It may occur following pneumonia,[33,34] lung transplantation,[35] migration of ascaris larvae,[36] poisoning,[37] and trauma.[38] Symptoms and signs are variable. Paroxysmal or persistent cough, hemoptysis, recurrent pneumonia, hypoxemia, and chest pain have been reported.[34,38] The definitive diagnosis is made by lung biopsy.

In summary, persistent hepatization of a lobe of the lung should be suspected in any patient with a history of trauma to the diaphragm and in patients with a persistent, chronic, and paroxysmal cough.

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Antihistamines and Asthma

Hugh S Mathewson MD

The bronchoconstrictor action of histamine has been well recognized since the basic researches of Dale and Laidlaw in 1910.[1] The release of histamine from tissue cells as part of the antigen-antibody reaction had been established by 1927,[2] and the accompanying bronchospasm demonstrated to be histamine-induced. Provocation of an asthmatic episode by administration of histamine was specifically described in 1932.[3] When antihistamines (H-1-antagonists) were developed by Bovet and associates in the 1940s,[4] experimental evidence pointed toward the value of these compounds in the treatment of bronchospastic states, and clinicians embarked confidently on their use in asthmatic patients.

The results were disappointing. Although the response to antihistamines in allergic rhinitis was strikingly beneficial, smooth muscle spasm in the lower airways was not often relieved, even when the drugs were given for extended periods.[5,6] Increasing the dosage only served to accentuate undesirable sedative and antimuscarinic side effects. By 1990 it was generally agreed that for relief of asthma attacks antihistamines were "singularly ineffectual."[7] It was recognized that other bronchoactive substances were released in antigen-antibody reactions, and the focus on mast cell degranulation confirmed that histamine was but one contributor to the bronchospastic episode. Histamine was primarily identified with the early asthmatic response, while lipoidal substances (leukotrienes, prostaglandins, platelet activating factor) and polypeptides (cytokines and others) showed more sustained activity and accounted for late asthmatic responses. Researches are now conducted on a broad spectrum of bronchoactive compounds associated with allergy and inflammation.

However, histamine has been the subject of continuing investigation throughout the latter half of this century. Three distinct classes of histamine receptors have been described. The first to be discovered, designated H-1,[8] are blocked by the compounds often called antihistamines. (Some proprietary products containing H-1 and H-2 receptor antagonists are shown in Table 1.) The second group of receptors (H-2) are associated with the gastric secretogogue activity of histamine;[9] H-2-blocking agents are utilized for treatment of hypersecretory states such as peptic ulcer disease. A wide structural difference and little overlap between their pharmacologic properties exists between H-1- and H-2-blockers. With respect to asthma therapy, there is nothing further to report concerning H-2-antagonists. A third receptor type (H-3) has been found in brain tissues.[10] Stimulation of H-3-receptors, which are presynaptic, initiates a negative feedback mechanism that inhibits histamine synthesis. There is

Table 1. Compounds with H-1- and H-2-Receptor Antagonist Properties and Proprietary Products in which They Are Found

Compound	Proprietary Products
H-1-Receptor Antagonists	
First generation	
diphenhydramine	Benadryl
tripelennamine	PBZ
chlorpheniramine	many over-the-counter 'cold' and allergy preparations
promethazine	Phenergan
hydroxyzine	Atarax, Vistaril
Second generation	
terfenadine	Seldane
astemizole	Hismanal
loratadine	Claritin
H-2-Receptor Antagonists	
cimetidine	Tagamet
ranitidine	Zantac
famotidine	Pepcid

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considerable interest in the development of H-3-active compounds, but as yet none of clinical value has appeared.

There may be reason to continue the study of H-1-blocking drugs for possible therapeutic applications in asthmatic patients. This subject has been addressed in a recent review by Bardin and Holgate.[11] The studies they report were nearly all conducted outside the USA. Several issues were raised that may yet indicate that H-1-antagonists of more recent design may offer future clinical promise.

In early clinical studies, a minor percentage of asthmatic patients reported beneficial effects from the antihistamines then available. Diphenhydramine, tripelennamine, promethazine, and chlorpheniramine, each a representative of a different chemical class of H-1-antagonists, all appeared to reduce asthmatic symptoms in up to 25% of patients.[5] Although these were mostly uncontrolled studies with little objective data, these first-generation antihistamines were apparently not "singularly ineffectual," even allowing for placebo effects and possibly some observational biases. It is common knowledge among allergists that an occasional patient taking an antihistamine for allergic rhinitis will notice a reduction in chest tightness and wheezing. The beneficial effects were, however, dose-related, and the antimuscarinic mucosal drying effects could be detrimental. More objectionable were unwanted gastrointestinal symptoms and sedative-hypnotic effects that interfered with daily activities.

The development of relatively non-sedating H-1-receptor antagonists in the 1970s provided a somewhat greater latitude in dosage compared to the older compounds. The more recent derivatives are chemically akin to the first-generation antihistamines, but are characterized by a lack of central nervous system penetration. Brain tissue concentrations are much reduced, but lung level values are undiminished. I briefly discuss five of these compounds: terfenadine, astemizole, cetirizine, azelastine, and mequitazine—although only the first two are approved for use in the USA. These are not new drugs; all were in clinical use before 1990.[12] Each has distinctive properties that offer potential benefits in the treatment of histamine-induced bronchospasm.

In atopic asthmatic subjects, Holgate and colleagues[13-15] reported that oral doses of terfenadine (180 mg), astemizole (10 mg), and azelastine (8.8 mg) provided 35-fold, 17-fold, and 50-fold protection, respectively, against histamine-induced bronchoconstriction. They estimated that about 50% of the early asthmatic response is contributed by histamine, the remainder from other mediators. Terfenadine has been shown to attenuate the bronchoconstrictor response to exercise.[16] Protection against late-phase responses is more controversial, although the Holgate group found that azelastine (8.8 mg) completely inhibited the late-phase reaction to inhaled allergen.[17]

Cetirizine is the major metabolite of hydroxyzine,[18] an antihistamine that was once widely used as an anxiolytic and minor tranquilizer. It is of interest because it not only blocks H-1-receptors,[19] but also prevents release of prostaglandin D_2 and platelet-activating factor, which contribute to bronchial inflammation.[20] Similarly, mequitazine, a phenothiazine related to promethazine, has been shown to inhibit the action of calmodulin and to interfere with mobilization of membrane phospholipids through inhibition of phospholipase A_2.[21] The latter is a characteristic anti-inflammatory action of corticosteroids. Thus, the effects of some H-1-antagonist drugs extend beyond histamine-receptor blockade and may occupy binding sites that prevent the actions of other bronchoactive transmitters and modulators.

An analogy can be drawn between the multiple blocking actions of some antihistamines and the properties of cromolyn-like compounds. A drug still available in Europe is ketotifen, which is usually classified as an antihistamine,[22] but is postulated also to inhibit mediator release from mast cells and to occupy receptors to bronchoactive compounds other than histamine. Ketotifen has been recommended for prophylaxis of asthma in much the same way that cromolyn and nedocromil are employed.[23]

Although H-1-receptor antagonists may have demonstrable effects in preventing asthma episodes, the question remains whether this is worthwhile, or whether these agents are of value for acute treatment. It is now generally agreed that the older antihistamines given orally to asthmatic patients in maximally tolerated doses over periods of several weeks have failed to demonstrate significant efficacy.[17] However, single-dose studies with terfenadine, azelastine, and cetirizine have demonstrated dose-related bronchodilation similar to that achieved with beta-agonist drugs.[24,25] Two recent studies have shown that terfenadine has bronchodilator action of possible therapeutic value.[26,27] The types of patients most likely to benefit from antihistamines are (1) young asthmatics; (2) seasonal pollen-induced asthma patients; and (3) severe asthma patients with reduced baseline airway caliber and presumed high basal histamine tone in the airways.

In 1995 an encyclopedic work on asthma and rhinitis—*New Medication for Rhinitis* appeared, whose chapters were written by authorities from the USA, UK, Scandinavia, and several other countries.[28] Whereas contributors to the work establish the role of H-1-antagonists in the treatment of allergic and persistent seasonal rhinitis beyond question, the statements on H-1-antagonist use in asthma are less enthusiastic. Terfenadine is recognized as occasionally effective in rescue medication of asthma episodes, although its failure to provide protection against methacholine challenge is also emphasized.[29] Other recent studies are cited that are indicative that antihistamines are "only of trivial use in chronic asthma."[30] However, it is restated by Simms[31] that patients with both allergic rhinitis and asthma are likely to obtain relief from lower airway symptoms when the newer long-acting H-1-blocking agents are employed.

It is probable that purely antihistaminic drugs will have no place in either the prophylaxis or treatment of asthma. However, the versatility of these molecules in occupying sites of other mediator receptors may lead to discovery of compounds of the antihistamine type that may be clinically beneficial—and, thus, the "singularly ineffectual" label may eventually be modified.

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The Role of Echocardiography in the Practice of Respiratory Care

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Introduction
Echocardiography: Technology & Techniques
The Normal Echocardiographic Examination
Assessing Ventricular Function
Assessing Systolic Function
Assessing Diastolic Function
Assessing Valvular Obstruction
Assessing Atrioventricular-Valve Insufficiency
The Role of Respiratory Care
In Summary

Introduction

As the care of patients in the critical care setting becomes more complex, an understanding of cardiovascular pathophysiology and the methods for its assessment become essential components of providing quality respiratory care. The complexity of delivering quality patient care and the expansion of the responsibilities of respiratory care practitioners (RCPs) in the critical care environment underscore the importance of a solid foundation in cardiovascular physiology and technology for RCPs. Cardiovascular anatomy and physiology play an important role in the respiratory care of critically ill patients, particularly infants with congenital heart disease. In infants and children with structural heart disease, the anatomy and, thus, the physiology may dictate the respiratory management. This becomes particularly important for the RCP because children with repaired and those with unrepaired congenital heart conditions are reaching adulthood in ever increasing numbers. In addition, the care of critically ill infants with primary pulmonary disease may be influenced by myocardial dysfunction.

The cardiovascular assessment of critically ill patients can be performed at the bedside using both noninvasive and invasive techniques. One of the more powerful noninvasive techniques is echocardiography. Using this technology, one can accurately elucidate the presence of anatomic and physiologic abnormalities in cardiovascular function that may affect the respiratory care of the patient. The tremendous advances in echocardiography coupled with the expanding role of the RCP in the intensive care unit have resulted in the need for RCPs to be aware of how echocardiography is used both to diagnose various pathophysiologic conditions and to develop therapeutic options. This review provides a basis for understanding the technical rudiments of echocardiography and its advantages and limitations and provides a foundation upon which RCPs can expand their knowledge of cardiovascular assessment.

Echocardiography: Technology & Techniques

Echocardiography has become a powerful tool for diagnosing and managing congenital and acquired heart disease. It is used for both critically ill patients in the intensive care unit and ambulatory patients. In essence, there are three parts to every echocardiographic examination whether it is performed from the chest wall or from the transesophageal approach—structure, or anatomy; dimensions; and physiology. The first echocardiographic technique is two-dimensional (2-D) imag-

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ing, which provides information about the structure and anatomy of the heart. In newborn infants, this is the most important examination because the diagnosis and management of these patients depend on an accurate anatomic diagnosis and not necessarily upon the physiology. A second technique used in echocardiography is M-mode (time-motion mode), which samples along a single ultrasound beam or line and, thus, allows higher resolution for measurements of chamber dimensions. Physiologic information can be obtained from the echocardiogram by using one of two Doppler techniques—the pulsed-Doppler examination, which allows the measurement of blood-flow velocities, and the color-Doppler examination, which facilitates the detection of abnormal blood-flow patterns within the heart and great vessels.

Cardiovascular abnormalities that affect the respiratory management of the patient generally fall into several categories. These include alterations in the function of the ventricles or in abnormalities of the semilunar or atrioventricular valves. Valvular abnormalities more specifically relate to stenoses or insufficiency of the valves. In this paper, we outline the normal echocardiographic examination including 2-D, M-mode, and Doppler echocardiography and review the echocardiographic techniques used to detect and measure abnormalities in cardiovascular function. Finally, we discuss the role of the RCP in the echocardiographic examination in the intensive care setting.

The Normal Echocardiographic Examination

2-D Echocardiography

The 2-D echocardiographic examination consists of obtaining images through several transthoracic echocardiographic windows. The echocardiographic windows are areas of soft tissue on the thorax and abdomen that permit the transmission of ultrasound without the significant attenuation that can occur when the ultrasound beam travels through bone. The parasternal window is located in the second or third intercostal space to the left of the sternum. The apical window is, as it sounds, located over the apex of the heart, at the point of maximal impulse of the heart; ie, at the point where the cardiac impulse can be felt. One of the most useful windows in pediatrics is the subcostal window, which is on the abdomen just inferior to the xiphoid process. In this view, the heart is imaged much like an antero-posterior or lateral chest radiograph. The final transthoracic view is through the suprasternal notch window, one can image the aortic arch and the pulmonary artery branches. The transesophageal window, with intraesophageal access, provides an unobstructed window from behind the heart and is an important advance in imaging those patients in whom transthoracic echocardiograms cannot be performed. This approach is particularly useful in adult patients.

In each of these windows, one can obtain a three-dimensional view by sweeping the transducer either from left to right or from an inferior to a superior position. The three-dimensional image, then, is really in the mind's eye, as one sweeps through a series of 2-D images. The third dimension is obtained by reconstructing this series of 2-D images. In this way, one can image the relationship of various structures to one another such as the location of a ventricular septal defect or of the great vessels.

In the parasternal view, if one aligns the transducer plane with the long-axis of the heart, then one can image the left-ventricular inflow and outflow at the same time (Fig. 1). In this view, one can see the left atrium, mitral valve, left ventricle, the left-ventricular outflow tract to the aorta, and the aortic valve. By sweeping the transducer to the right, one can image the tricuspid valve and the right-ventricular inflow tract. If the transducer is angled to the left from the left-ventricular view, one can image the pulmonary artery and the right-ventricular outflow tract. The parasternal long-axis view is useful in imaging ventricular septal defects and the great vessels and their spatial relationship to one another. An orthogonal view to the long axis is the short-axis parasternal view, which is obtained by rotating the transducer 90° in a clockwise direction. If the transducer is angled inferiorly, a cross-section of both the right and left ventricle is imaged (Fig. 2). This view provides an excellent image of the left ventricle for assessing left-ventricular function and wall-motion abnormalities.

Fig. 1. Parasternal long-axis view of the heart. In this view the left-ventricular inflow and outflow tracts are displayed. The left atrium (LA) can be seen posteriorly. The mitral valve is located between the left atrium and left ventricle. The left ventricle (LV) and the aorta (Ao) are seen in the middle portion of the picture and most anteriorly one can see the right ventricle (RV).

When the transducer is swept superiorly toward the base of the heart, the aortic valve can be imaged in cross-section in the middle of the screen (Fig. 3). In this view, the right-ventricular inflow with the tricuspid valve and the outflow portions with the pulmonary valve can be seen wrapping around

the aorta, the left and right atria appear to be posterior. The short-axis view at the level of the aortic valve is particularly good for imaging abnormalities of the aortic and pulmonary valves as well as the right-ventricular outflow tract. This is also an excellent window for imaging ventricular septal defects that are located in the outlet septum beneath the aortic and pulmonary valve.

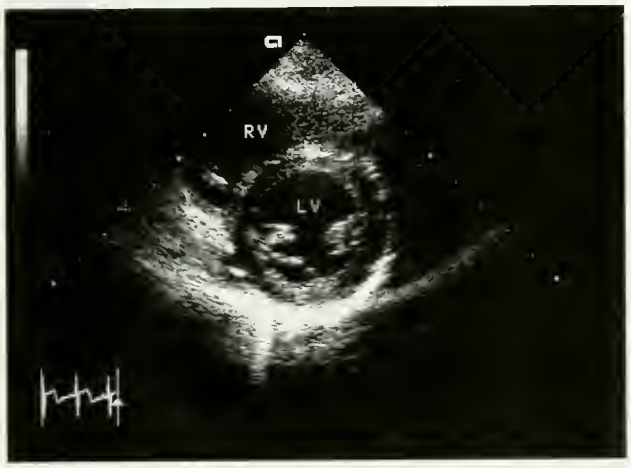

Fig. 2. Short-axis view of the left ventricle (LV) at the level of the papillary muscles. The right ventricle (RV) can be seen anterior and to the right of the left ventricle.

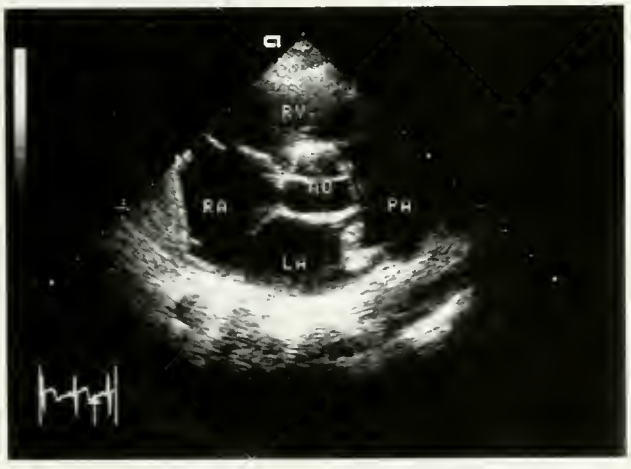

Fig. 3. Parasternal short-axis view at the level of the outflow tract. The right-ventricular (RV) inflow and outflow tracts can be seen sweeping around the aorta (Ao). Note that the pulmonary artery (PA) is wrapped around the aorta, which is seen in cross-section. RA = right atrium, LA = left atrium.

The apical 4-chamber view provides the most recognizable view of the heart because the 4 chambers of the heart can be seen in the same plane (Fig. 4). By sweeping posteriorly, the coronary sinus can be imaged. An anterior sweep brings the left-ventricular outflow tract and aorta into view. This view is best for imaging the mitral and tricuspid valves and any abnormalities that may be present. Aortic insufficiency is also often best imaged using color-Doppler from the apical 5-chamber view, which now includes the 4 chambers of the heart and the left-ventricular outflow tract.

Fig. 4. Apical 4-chamber view of the heart. This is at the inflow level of the heart, and the tricuspid and mitral valves are seen in the closed position. LA = left atrium, LV = left ventricle, RA = right atrium, RV= right ventricle.

The subcostal view provides the most information in complex congenital heart defects as one sweeps through all 3 portions of the heart (ie, the inflow portion, which is most posterior and inferior; the left-ventricular outflow portion of the heart, which is in the mid-portion of the heart; and the right-ventricular outflow tract, which is the most anterior portion of the heart). The subcostal sagittal view is similarly useful, except that one sweeps from left to right, imaging the heart as in a lateral chest film. In this manner, the heart is imaged from multiple views and the detailed anatomy of the heart can be delineated. Finally, the suprasternal-notch view is an excellent way to image the aortic arch and associated anomalies, such as coarctation of the aorta. This view is also particularly helpful in imaging the pulmonary artery branches and, in the coronal view, the pulmonary veins.

M-Mode Echocardiography

Whereas 2-D echocardiography uses multiple scan lines (dimensions) to display a 2-dimensional image of the heart, M-mode echocardiography uses a single scan line. This was the initial method available for echocardiography, and a great body of literature describes its usefulness as a diagnostic technique. With the advent of 2-D imaging, the persisting utility of M-mode echocardiography is in its resolution. When

multiple scan lines are used, as in 2-D echocardiography, time must be shared among the different scan lines to obtain the image, decreasing the resolution of the image. Because of its excellent resolution, M-mode echocardiography is used, primarily, to accurately measure chamber dimensions.

Doppler Echocardiography

The Doppler technique emerges from the basic principle that as an object moves towards or away from a stationary object the frequency of the sound from that object changes in direct proportion to its velocity. In this manner, Doppler echocardiography can measure the velocity and direction of blood flow within the heart. The velocity is calculated using the Doppler equation.

$$V = \frac{cf_d}{2f_o \cos\theta} \quad [1]$$

where c = the velocity of sound in human tissue, f_d = the observed Doppler frequency shift, f_o = the transmitted ultrasound frequency, and θ = the intercept angle between the ultrasound beam and the blood flow.

There are two types of Doppler echocardiography that allow the accurate measurement of blood-flow velocity. Pulsed Doppler samples the blood-flow velocity within a particular range gate (ie, within a specific region of the heart in what is essentially a tear-drop shape—Fig. 5). Although this allows the accurate measurement of blood-flow velocity in a well circumscribed region of the heart, it does not allow the measurement of high-velocity blood flow. To measure high-velocity blood flow, continuous-wave Doppler is used. In theory, this technique can measure an infinite velocity (Fig. 6). Its limitation lies in the fact that there is no range resolution, and the velocities are sampled along a continuous line—ie, the velocity measured may be taken from any point on the line that is displayed on the screen. This may result in some ambiguity as to where the maximal velocity shift is occurring; therefore, it is not helpful when the precise location of a pressure drop, or gradient, needs to be known, such as in a patient with serial obstructions—eg, valvular aortic stenosis and subvalvular aortic stenosis.

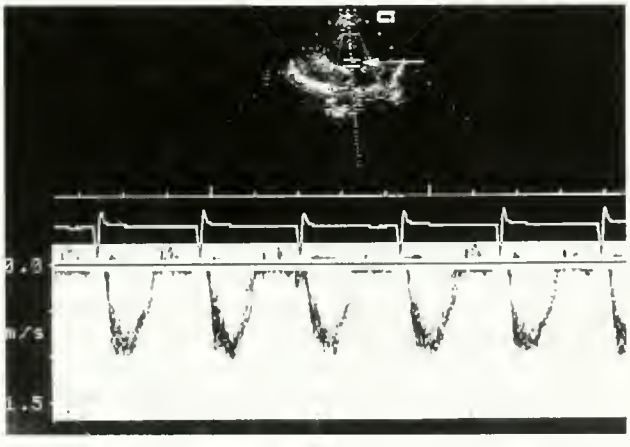

Fig. 5. Example of a pulsed-Doppler examination in the pulmonary-outflow tract. The sample volume (arrow) is indicated by the two horizontal lines located along the cursor. The Doppler spectral tracing at the bottom of the image shows laminar flow at approximately 1 m/s.

Fig. 6. Example of a continuous-wave Doppler tracing of turbulent blood flow in the ascending aorta in a patient with aortic stenosis. Note that in contrast to the pulsed-Doppler examination, which has laminar flow (Fig. 5), the Doppler signal is entirely filled in, indicating turbulent flow. The velocity in this tracing is 3 m/s indicating mild-to-moderate aortic stenosis. In this example, the flow is toward the transducer and is, therefore, displayed above the baseline.

A modification of the pulsed Doppler technique assigns color to the blood flow in multiple sample volumes within a box. The color is assigned based upon the direction and velocity of blood flow. By convention, flow towards the transducer is assigned the color red and flow away from the transducer is assigned the color blue. When there is turbulent or high-velocity flow, a mosaic of colors is often seen (Fig. 7). Although color Doppler does not allow the quantification of blood flow velocity, it does provide qualitative information on locations of abnormal or disturbed flow. For instance, a patent ductus arteriosus can be readily detected using color-Doppler flow-mapping from the parasternal short-axis view from which turbulent flow can be seen from the aorta into the pulmonary artery. The ductus jet can usually be seen along the lateral wall of the pulmonary artery.

Assessing Ventricular Function

Whatever the etiology, abnormalities in ventricular function, both systolic and diastolic, can occur in patients with cardiorespiratory disease and complicate the patient's course. Evaluation of ventricular function in the intensive care setting can be very difficult as most indices of systolic function depend on preload, afterload, and contractility. Therefore, all measurements of ventricular function must be evaluated with these limitations in mind. The primary noninvasive method

Fig. 7. Parasternal long-axis view of the left-ventricular outflow tract in a patient with aortic stenosis. The pure blue, laminar flow is seen at the level of the left-ventricular outflow tract. The turbulent flow displayed in this view as a mosaic of colors begins at the level of the aortic valve. LA = left atrium, LV= left ventricle, RV= right ventricle.

for evaluating ventricular function in the intensive care setting is 2-D echocardiography. The greatest advantage of this technique is that it can be performed at the bedside, eliminating the need to transport critically ill patients to other areas of the hospital. The obvious advantage is that the risk for accidental extubation and subsequent atelectasis and loss of functional residual capacity is reduced.

Assessing Systolic Function

The evaluation of systolic function is directed at determining the adequacy of ventricular contraction. All methods for evaluating systolic function are dependent on both preload and afterload and, therefore, may not truly reflect the heart's contraction characteristics. The two methods used to evaluate systolic function are M-mode and 2-D echocardiography. M-mode is used to measure the shortening fraction. Two-dimensional echocardiography may provide an indication as to whether regional wall-motion abnormalities are apparent from the parasternal short-axis window or the apical 4-chamber view. This may help determine the mechanism and location of the myocardial insult. In addition, 2-D echocardiographic measurements allow the calculation of ejection fraction. Doppler echocardiography can be used to measure the velocity of fiber shortening.

M-Mode Echocardiography

M-mode echocardiography provides a single scan line of information at a much higher frame rate than can be obtained by 2-D echocardiography. This allows the accurate measurement of ventricular dimensions and, thus, the percent ventricular shortening fraction (%SF).

$$\%SF = \frac{LVEDD - LVESD}{LVEDD} \times 100, \quad [2]$$

where LVEDD = left-ventricular end-diastolic diameter (cm) and LVESD = left-ventricular end-systolic diameter (cm).

The normal range for %SF is 28-42%. However, because M-mode echocardiography provides only a single line of information, it is not reliable when wall-motion abnormalities are present or in the assessment of ventricles with abnormal shapes. One example of this limitation is in patients with regional abnormalities of ventricular function, which may occur after myocardial infarction. In this condition, portions of the myocardium that suffer an infarction may have dramatically altered motion, whereas other components of the ventricle may function more normally. As a result, if the M-mode beam cuts through the portion of the ventricle that is normally functioning, an erroneous value for shortening fraction results. These limitations of M-mode have stimulated research into several other echocardiographic methods of evaluating ventricular function. Again, the shortening fraction depends on preload, afterload, and contractility, so that a decrease in shortening fraction may not necessarily represent a decrease in stroke power.

2-D Echocardiography

In most instances, the left ventricle can be adequately imaged so that the volumes in the ventricle at end-systole and end-diastole can be measured. This allows calculation of an ejection fraction (EF),

$$EF = \frac{LVEDV - LVESV}{LVEDV}, \quad [3]$$

where LVEDV = left-ventricular end-diastolic volume, and LVESV = left-ventricular end-systolic volume.

Although many methods are available for the calculation of volumes and ejection fraction, the modified Simpson's rule is the method most widely used and accepted. This method calculates the volume of the ventricle by dividing it into 20 equal cylinders and summing their volume. This complex process is now performed automatically by many echocardiographic computer programs after the endocardial borders of the ventricle in systole and diastole have been traced from the apical 4-chamber view. Results obtained in this manner closely correlate with the EF derived by cardiac catheterization. The normal EF is 65-75%. The EF is also an ejection phase index that depends on preload and afterload, so that a decrease in the EF may not necessarily indicate a decrease in the strength of contraction.

Doppler Echocardiography

Another frequently utilized index of ventricular function is the velocity of circumferential-fiber shortening (VCF).

This is calculated by taking the shortening fraction (Equation 2) and dividing it by the left ventricular ejection time. The left ventricular ejection time is measured from the Doppler spectral tracing of the left-ventricular outflow, at the level of the aortic annulus. In this way, the afterload of the ventricle is taken into account. In addition, the VCF can be corrected (VCF_c) for the heart rate if the left-ventricular ejection time is divided by the square root of the duration of R-R interval.

$$VCF_c = \frac{(LVEDD - LVESD)/LVEDD}{\text{LV ejection time}/\sqrt{R - R}} , \quad [4]$$

where LVEDD = left-ventricular end-diastolic diameter (cm), LVESD = left-ventricular end-systolic diameter (cm), and R – R = the time (s) of a complete cardiac cycle.

The normal value for the VCF_c is 0.97 ± 0.07.[1] Although this measurement is not a completely load-independent measure of ventricular function, it is easily performed at the bedside and is, therefore, useful in the assessment of ventricular function.

Assessing Diastolic Function

Abnormalities in diastolic function have recently become recognized as an early indication of decreased ventricular function because they may be evident or manifest themselves before abnormalities in systolic function can be seen. As delineated in a previous paper,[2] diastole is an energy-requiring process. Calcium ions must be pumped against a gradient into the sarcoplasmic reticulum. In addition, myosin cross-bridge detachment requires the hydrolysis of an adenosine triphosphate molecule. Therefore, the assessment of diastolic dysfunction may be a more sensitive indicator of myocardial dysfunction, and diastolic assessment has become an important method of evaluating ventricular function. Diastolic abnormalities of the left ventricle have been correlated with the mitral-valve-Doppler inflow velocities.[3] Although the mitral-valve-Doppler inflow patterns reflect the pressure difference, or gradient, across the mitral valve during diastole and are not specific for a given disease state, they have correlated well with catheterization-derived measurements of ventricular function during diastole.

Assessing Valvular Obstruction

Obstruction to right or left ventricular outflow or inflow may result in substantial hemodynamic and respiratory alterations in patients who require intensive care. For example, marked obstruction of left-ventricular inflow or outflow may result in passive congestion of the lungs and, thus, alter respiratory mechanics. Therefore, the echocardiographic assessment of patients with valvular obstruction involves not only identifying the level of the obstruction but also determining its severity. Two-dimensional echocardiography is the best method of identifying the level of the obstruction because the anatomy can be directly visualized. Color Doppler may aid in identifying the level of the obstruction by showing points of turbulence (Fig. 7). Using echocardiography, one is able not only to identify the level of the obstruction but also the nature of the obstruction—eg, in subvalvular obstruction, a determination can be made as to whether a fixed or dynamic obstruction is present. Differentiating between a fixed and dynamic obstruction may have important implications for the hemodynamic and respiratory management of the patient.

The severity of the obstruction can only be ascertained by measuring the magnitude of the obstruction (ie, the pressure gradient across the obstruction) using Doppler echocardiography. The simplified Bernoulli equation provides a rapid and accurate assessment of the severity of the obstruction.

$$P_1 - P_2 = 4V^2 \quad [5]$$

where P_1 and P_2 = pressures on either side of the obstruction, and V = blood-flow velocity across it.

However, in the critically ill patient with low cardiac output, the gradient ($P_1 - P_2$) may not truly reflect the severity of the stenosis because it depends on the flow across the valve. For example, in patients with a very low cardiac output after myocardial infarction, there may not be a large gradient, despite the presence of severe or even critical obstruction. In such circumstances, other measurements such as the mean gradient or valve area should be calculated. The mean gradient is obtained by tracing the outer envelope of the Doppler spectral tracing on-line or on an off-line analysis system and averaging all the instantaneous gradients throughout systole. The valve area can be calculated using the continuity equation.

$$A_1 \cdot V_1 = A_2 \cdot V_2, \quad [6]$$

where A = cross-sectional area of the lumen proximal to the valve (A_1) and of the stenotic orifice (A_2), V_1 = mean velocity proximal to the valve, and V_2 = mean velocity of the greatest of all the spectral signals across the stenosis.

In aortic-valve stenosis, the cross-sectional area from inner edge to inner edge (the flow area) proximal to the stenosis can be measured from the long-axis view at the level of the valve leaflets during early systole. This calculation assumes the area to be circular and is calculated using

$$A = \frac{\pi d^2}{4} , \quad [7]$$

where A = area, and d = diameter.

The velocity proximal to the stenosis (V_1) can be measured from the apical window using pulsed Doppler in the left-ventricular outflow tract, and the maximal velocity (V_2) can be measured with continuous-wave Doppler. With these measurements, the valve area (A_2) can be calculated. The assessment of valve area is an essential component of the cardiovascular evaluation and can help determine whether surgical intervention is required. The limitations of the technique rest on the assumption that the left ventricular outflow tract is circular and that the proximal and maximal velocity are accurately measured because these numbers are squared in the Bernoulli equation, compounding the error posed by the inaccurate measurement.

Assessing Atrioventricular-Valve Insufficiency

Atrioventricular-valve insufficiency often complicates the recovery from critical illness of adult and pediatric patients. Insufficiency of the mitral valve can cause volume overload of the left ventricle and passive congestion in the pulmonary capillary bed, resulting in a prolonged ventilatory course or in a failure to wean from the ventilator. In contrast, insufficiency of the tricuspid valve results in hepatic congestion and decreased cardiac output. Determining the severity of atrioventricular-valve insufficiency can be difficult, and information derived from the diagnostic studies must be correlated with the patient's clinical course.

Two-dimensional and color-Doppler echocardiography can provide important information on the location of the insufficiency. The anatomic location and nature of the insufficiency helps not only to determine the need for surgical repair but also to determine the type of repair that can be performed—valvuloplasty versus valve replacement. Although it is tempting to use the area of the color-Doppler flow jet, it does not reflect the degree of insufficiency and is more related to the direction of the jet, the compliance of the atrium, and technical factors, such as the settings on the ultrasound machine that determine how data are acquired and displayed.[4,5] The size of the jet at the regurgitant orifice is probably the best indicator of the severity of valvular incompetence (Fig. 8). The size of the left atrium and left ventricle in mitral insufficiency and the right atrium and right ventricle in tricuspid insufficiency may not be reliable indicators of the severity of the insufficiency in the postoperative patient because they may be influenced by preoperative volume overload of the chambers.

The Role of Respiratory Care

As the role of RCPs in the intensive care setting increases, it is only natural that they will need not only to respond to the findings on new diagnostic modalities such as echocardiography, but also to participate in the examination as part of the patient care team. In many instances, the echocardiographic

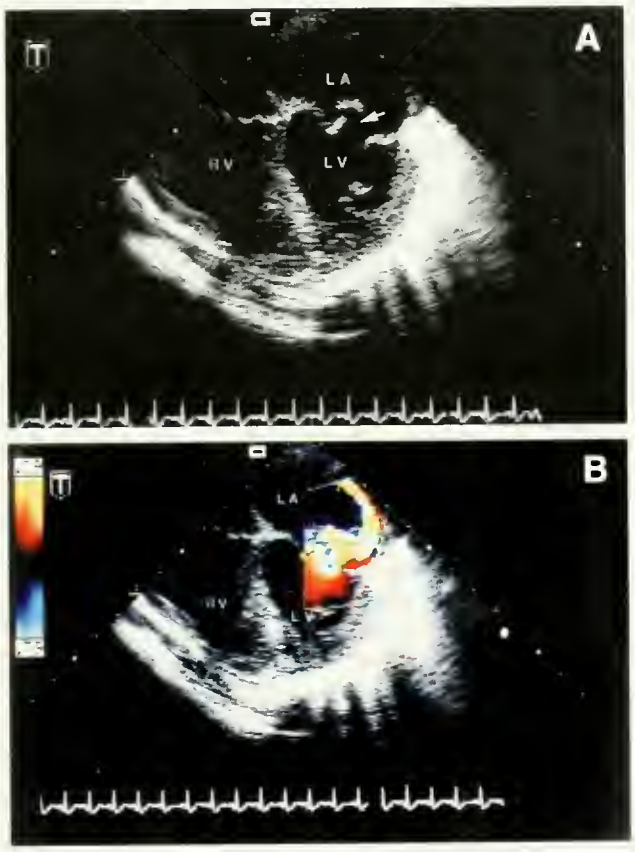

Fig. 8. (A) Transesophageal two-dimensional echocardiographic view of the 4-chambers of the heart. The transducer in this image is located posterior to the left atrium. This is an example of a patient with a flail mitral valve and its large regurgitant orifice (arrow). (B) Color-Doppler examination of the same patient with the flail mitral valve showing the large regurgitant color orifice at the level of the valve. Note that the color jet layers out along the left atrial wall.

examination has respiratory care implications. One situation that requires particular interaction is in transesophageal echocardiograms performed in the intensive care unit. During transesophageal echocardiography, sedation is frequently required. The administration of sedating agents requires vigorous cardiorespiratory monitoring because these patients have limited cardiorespiratory reserve and are sensitive to minor alterations in cardiorespiratory performance. Therefore, the administration of sedation may decrease cardiac or respiratory function and result in a decrease in oxygen delivery. Sedation can lead to a decrease in respiratory effort with a loss of functional residual capacity and atelectasis and a reduction in gas exchange. For these reasons, some patients may require intubation prior to or during transesophageal echocardiography to adequately examine the cardiovascular system. In patients who are already intubated, transesophageal echocardiography may result in respiratory destabilization secondary to displacement, advancement, kinking, and/or obstruction of the endotracheal tube.

Another group of patients that requires optimal involvement of the RCP is small infants and children in the intensive care unit. These patients are especially sensitive to minor alterations in cardiorespiratory function because they have limited ability to increase their cardiac output or respiratory effort in response to increased demands. In these patients, the echocardiographic examination can be a destabilizing event, and reductions in cardiac output and oxyhemoglobin saturation occur frequently. When these patients become unstable during an echocardiogram, it usually is a direct result of the effects of the sedation that is administered for the examination. Sedatives can cause a profound decrease in minute ventilation resulting in atelectasis and alterations in gas exchange, as previously described. When minute ventilation decreases in response to the reduction of the patient's respiratory efforts, a reduction in gas exchange follows, if the patient's ventilator settings are not adjusted. Such destabilization can be avoided by evaluating the patient prior to the procedure to determine whether risk of respiratory compromise is present and by continuous assessment of the patient's minute ventilation and respiratory rate during the procedure. Patients whose spontaneous respiratory rate is high prior to the echocardiogram are likely to destabilize during the echocardiogram. If the patient's respiratory effort decreases during the study, the settings on the ventilator should be increased to provide adequate respiratory support until spontaneous efforts return to appropriate levels.

Compression of the pulmonary parenchyma can occur as a result of compression of the thorax by transthoracic echocardiography or displacement of the diaphragm superiorly from transabdominal echocardiography. These two instances result in a 'functional' reduction in pulmonary compliance, functional residual capacity, and alveolar expansion. The consequences are an increase in peak inspiratory pressure during volume-control ventilation or a reduction in tidal volume during pressure-control ventilation that may lead to a dramatic reduction in gas exchange and/or pulmonary barotrauma. The RCP should anticipate these events during the procedure and set the ventilator-alarm limits so that alterations in functional pulmonary compliance can be detected. These limits include peak-inspiratory pressure, mean airway pressure, and tidal volume. If abnormalities are detected during echocardiography, the RCP should suggest that the study be temporarily interrupted until the patient recovers. A well-versed RCP keeps the physicians informed about the changes in compliance, peak-inspiratory pressure, mean airway pressure, and tidal volume that have resulted from these reductions in functional compliance. In this manner the RCP can help prevent destabilizing events during the echocardiographic evaluation.

In addition to participating in the monitoring of patients undergoing echocardiography, the RCP may participate directly in the use of echocardiography to manage patients. One example is the use of echocardiography to determine the most efficient level of positive end-expiratory pressure or 'best PEEP.' Best PEEP is that which optimizes oxygen delivery by increasing oxygen content while maintaining cardiac output and diastolic function.[6] It is determined by measuring cardiac function using echocardiography as the PEEP level is incrementally increased, then decreased. Cardiac output can be determined from the continuity equation (Equation 6). Stroke volume can be calculated by

$$SV = \frac{CSA \cdot V\,(R - R)}{1000\ cm^3/L}, \qquad [8]$$

where CSA = the cross-sectional area of the valve across which the flow is occurring (cm^2), V = mean velocity across the valve (cm/s), and R – R = the duration of a cardiac cycle (s).

To calculate the cardiac output, one need only multiply the stroke volume by the heart rate.[7,8] The aortic valve is the most reliable site for measuring the cardiac output because the annulus is rigid and does not change appreciably during the cardiac cycle. Once the annulus has been measured, then one only needs to obtain the Doppler flow velocity from the apical window for each PEEP setting. Because the aortic annulus is essentially rigid, the changes in cardiac output are directly proportional to the integral of the Doppler spectral tracing.

In Summary

The changing critical care environment provides a wonderful opportunity for RCPs to expand their influence in the critical care arena. We hope that a better understanding of one of the new advances in critical care diagnostic techniques, echocardiography, challenges RCPs to increase their input into this important discipline of critical care.

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Glossary for Echocardiography in Respiratory Care

adenosine triphosphate: Adenosine triphosphate (ATP) donates its terminal energy-rich phosphate group to any of a number of acceptor molecules, enabling them to take place in subsequent enzyme-catalyzed biochemical reactions—oxidative phosphorylation. In myocytes, it is the formation of high-energy phosphoric bonds (eg, pyrophosphates) from the energy released by the dehydrogenation (ie, oxidation) of various substrates—protein kinases in the sarcoplasm of muscle cells phosphorylate myosin, ATPase, and calcium channels—to cause increased myocardial contractility.

afterload: the 'downstream' resistance imposed on a ventricle. The afterload of the right ventricle is the force opposing ejection caused by the resistance of the pulmonary vasculature; whereas, the afterload of the left ventricle is the force opposing ejection caused by the resistance of the systemic circulation. Reducing afterload on an impaired ventricle improve its function. Afterload was once considered to be the arterial pressure or some other measure of the force that a ventricle must overcome while it contracts during systolic ejection, contributed to by aortic or pulmonic artery impedance, peripheral vascular resistance, and mass and viscosity of blood. It is now more rigorously expressed in terms of the wall stress—ie, the tension per unit cross-sectional area in the ventricular muscle fibers (calculated by Laplace's law from internal radius and wall thickness) that is required to produce the transmural (across the heart) pressure required for systolic ejection.

annulus: the fibrous ring that supports the valve tissue in the heart.

aortic stenosis: pathologic narrowing of the aortic valve orifice.

apical window: see **echocardiographic window**.

atrioventricular valve: the heart valve that separates the atria and the ventricles—open during diastole to allow the blood to enter the ventricle and closed during ventricular systole to prevent the blood from leaking back (regurgitating) into the atrium.

Bernoulli equation: describes the relationship between the flow velocity across a narrowed vessel or valve and the pressure drop that occurs.

$$P_1 - P_2 = 4V^2$$

where P_1 and P_2 = pressures on either side of the obstruction, and V = blood-flow velocity across it.

'best' PEEP: the end-expiratory pressure that maximizes oxygen delivery—maximizes lung volume recruitment while maintaining adequate cardiac output.

cardiac output: the amount of blood ejected by the heart in a unit of time, usually expressed in L/min. It is the product of the stroke volume (L/beat) and heart rate (beats/min):

$$\frac{\text{volume(L)}}{\text{beat}} \times \frac{\text{beats}}{\text{min}} = \frac{\text{volume(L)}}{\text{min}}.$$

coarctation of the aorta: a **congenital heart defect** that causes obstruction to flow in the descending aorta. The obstruction is caused by a fibrous shelf that protrudes into the lumen of the aorta.

color Doppler: the ultrasound machine assigns a color to the moving red blood cells based upon the direction and velocity of flow. By definition, red flow is towards the transducer and blue flow is away from the transducer. Its advantage is that blood flow patterns can be seen within the heart but its disadvantage is that quantitative measurements cannot be made as they are in **pulsed** and **continuous-wave Doppler**.

congenital heart defect: existing at birth; refers to anomalies of the cardiovascular system, which may be either hered-

itary (genetic) or influenced by events occurring during gestation, up to the moment of birth.

continuous-wave Doppler: used to measure blood-flow velocities in the heart and blood vessels. In theory, this technique permits the measurement of infinite velocity but cannot localize precisely where that velocity is occurring in the heart.

contractility: inotropic state of the myocardium; the ability or property of muscle to shorten or develop increased tension. A term used to describe the intrinsic properties of the muscle not related to **preload** or **afterload**. As long as heart rate, **preload**, and **afterload** do not change, an increase in contractility because of endogenous or exogenous catecholamines results in an increase in the force of contraction, myocardial oxygen uptake, and cytoplasmic calcium concentration.

coronal view: the echocardiographic plane that transects the heart in a superior to inferior and a left to right orientation. If one can picture walking directly through a mirror, the orientation of the mirror when you are half way through would be the coronal plane of the body and, thus, the heart.

diastolic ventricular function: ventricular function during the period during which the left atrium empties into the left ventricle. Diastole begins when the aortic valve (between the left ventricle and aorta) closes and ends when systole begins with the closing of the mitral valve (between the left atrium and ventricle). It alternates rhythmically with systole or contraction of the heart.

Doppler: the physical principle described by Johan Doppler that the frequency of a sound wave changes relative to the velocity of an object as it moves towards or away from an observer. In echocardiography, the 'observer' is the transducer, which emits and receives a sound wave at a given frequency. The object is the red blood cells as it moves through the heart and blood vessels. As red blood cells move towards or away from the transducer, the sound wave that returns to the transducer, after hitting the red blood cells, has a different frequency, which is proportional to the velocity of the blood cells. **Pulsed Doppler** allows the velocity to be measured in a specific area of the heart but is limited by not being able to measure high velocity flow. In theory, **continuous-wave Doppler** permits the measurement of infinite velocity but cannot localize precisely where it is occurring in the heart.

ductus arteriosus: a vessel that connects the pulmonary artery to the aorta and allows the blood to bypass the fluid-filled lungs in utero. In normal, term infants, the PDA closes spontaneously soon after birth.

echocardiographic window: an area of soft tissue on the chest or abdomen that allows a view of the heart that is not obstructed by ribs or lung. There are four standard views; the first is along the sternum in the second or third intercostal space (**parasternal window**) and includes the long-axis and the orthogonal (perpendicular) **short-axis view**; the second is the **apical window** which is obtained by placing the transducer over the apex of the heart, which is at the point of maximal impulse; the subcostal or subxiphoid view is obtained by placing the transducer in the abdomen just below the sternum; the final view is the **suprasternal notch view**, obtained by placing the transducer above the sternal notch.

echocardiography: ultrasound evaluation of the heart. Sound energy is transmitted into the body and reflected back to the transducer by the encountered tissues. Deflections or images appear on the monitor.

ejection fraction, systolic: The ejection fraction is that fraction of the end-diastolic volume that is ejected during systole, ie, the stroke volume divided by end-diastolic volume, normally 0.75-0.80, ie, 75-80%. With the onset of congestive heart failure, the ejection fraction decreases, sometimes to as little as 0.10 (10%), in severe cases.

frame rate: the echocardiographic image that is displayed on the screen is a composite of many still images. The number of images displayed per second is the frame rate.

long-axis view: see **echocardiographic window.**

M-mode (time-motion mode): the first form of clinically useful ultrasound. It uses a single scan line rather than the multiple scan lines, used to develop the two-dimensional image. Its high resolution enables highly accurate measurement of valvular and chamber dimensions and is its greatest advantage.

mitral valve: the **atrioventricular valve** between the left atrium and left ventricle.

myosin: one of the proteins of muscle fiber essential for muscle contraction. Its binding to actin and subsequent conformational change results in muscle-fiber shortening known as muscle contraction.

orthogonal view: See **echocardiographic window.**

outlet septum: the portion of the ventricular septum that lies between the aortic and pulmonary valves.

parasternal window: See **echocardiographic window.**

preload: volume of blood in the heart at the start of contraction. It is normally estimated using end-diastolic pressure and varies inversely with intrathoracic pressure. Under normal cir-

cumstances, larger preload results in greater ventricular stretching and larger stroke volume. The amount of stretching the ventricle undergoes prior to contraction. It is estimated by the measurement of end-diastolic ventricular volume. In the intact heart, the stretch is proportional to the volume of venous return delivered to the ventricle during diastole.

Pulsed Doppler: An echocardiographic technique that allows blood-flow velocity to be measured in a specific area of the heart but is limited by an inability to measure high-velocity flow.

R-R interval: the time between two consecutive R waves on the electrocardiogram—the duration of one cycle (beat) of the heart.

sagittal view: the echocardiographic plane that transects the heart in a superior to inferior and anterior to posterior plane. This plane is through the body in a way that divides the body in half—a cross-section.

sarcoplasmic reticulum: multiple folded membranes within the myocyte (muscle cell); the site of calcium storage and release.

scan line: The ultrasound beam is made up of multiple lines, which make up the two-dimensional echocardiographic image. The transducer contains 128 elements, which send and receive sound waves. Each element has its own scan line.

semilunar valve: the aortic and pulmonary valves located between the ventricles and the great vessels (aorta and pulmonary artery). They open to allow flow out to the body (aortic valve) and lungs (pulmonic valve) during ventricular contraction and close when the ventricle is filling during diastole to prevent the blood from leaking back into the ventricles (regurgitating) from the great vessels.

short-axis view: See **echocardiographic window**.

shortening fraction: the percent of change between the systolic and diastolic dimension of the heart, used as an index of left-ventricular strength.

$$\%SF = \frac{LVEDD - LVESD}{LVEDD} \times 100,$$

where LVEDD = left-ventricular end-diastolic diameter (cm) and LVESD = left-ventricular end-systolic diameter (cm).

Simpson's rule: a method used to calculate the volume of the left ventricle and, then, **ejection fraction**. This method assumes that the ventricle is an ellipsoid that is divided into 20 stacked disks. The volumes of the individual disks are calculated and summed.

spectral tracing: the graphic display of the Doppler signal, with time along the horizontal axis and velocity along the vertical axis.

stroke volume: the volume of blood ejected from the heart with each contraction, expressed in liters. It can be estimated by

$$SV = \frac{CSA \cdot V\,(R - R)}{1000\ cm^3/L},$$

where CSA = the cross-sectional area of the valve across which the flow is occurring (cm^2), V = mean velocity across the valve (cm/s), and R – R = the duration of a cardiac cycle (s).

subcostal window: See **echocardiographic window**.

suprasternal notch window: See **echocardiographic window**.

systolic ventricular function: function during systole—the phase of the heart beat during which the ventricles contract. In the left ventricle, it is the period that begins with the closure of the mitral valve and ends with the closure of the aortic valve.

three-dimensional view: the view that is obtained by summing the **two-dimensional images**.

transducer plane: the two-dimensional fan-shaped plane that is sent out from the transducer and is usually oriented along the length of the transducer.

transesophageal window: A specialized ultrasound transducer is placed into the esophagus to obtain images from behind the heart. This image is superior to those obtained through other windows, particularly in adults, because the heart structures are closer to the transducer and there is no interference from ribs or lungs.

tricuspid valve: the **atrioventricular valve** that is located between the right atrium and the right ventricle.

valvuloplasty: the use of a balloon catheter or incision to open up a narrowed valve.

ventricular septal defect: a defect or hole in the wall between the left and right ventricle, allowing blood to flow between the two chambers.

Severe Cardiorespiratory Dysfunction following Cold-Water Submersion: The Successful Use of Extracorporeal Membrane Oxygenation

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Introduction

Drowning is the second most common cause of accidental death and the third leading cause of all mortality in children.[1-3] Approximately 8,700 drownings occur annually in the United States.[4] It is estimated that there are five times more near-drowning accidents than drownings, with near-drowning defined as survival from a submersion accident beyond 24 hours.[4,5] Overall survival rates for submersion accidents have been reported to be 66%; whereas, overall survival with good neurologic recovery is estimated to be 56%.[4,6]

Morbidity and mortality secondary to pulmonary dysfunction are common following submersion accidents.[2,4,7,8] Both fresh- and saltwater aspiration are equally likely to result in surfactant deactivation and damage to the alveolar basement membrane.[2,9] In both conditions, alveolitis and pulmonary edema occur. Acute respiratory distress syndrome (ARDS) was reported in 27% of near-drowning patients in a series by Biggart and Bohn.[6] Only 20% of these patients with ARDS survived with intact neurologic function. Levin et al[4] state that intrapulmonary shunting can increase from the normal 5-18% to 75% after a submersion accident. In contrast to pulmonary failure, cardiac failure after a submersion is uncommon,[4,10,11] and the development of combined cardiorespiratory failure is extremely rare.

Despite the high morbidity and mortality in submersion victims often associated with pulmonary dysfunction and occasionally associated with cardiac dysfunction, there has been only a single prior report in which extracorporeal membrane oxygenation (ECMO) was used for pulmonary failure after a submersion incident.[12] In that patient, cardiovascular dysfunction was not prominent. Our case report illustrates the successful use of ECMO in the treatment of severe combined pulmonary and cardiac dysfunction following prolonged cold-water submersion.

Case Report

A healthy, 3-year-old, 15-kg boy was a passenger in a 4-wheel, all-terrain vehicle when the driver missed a turn and the vehicle slid into a partially frozen, freshwater pond. The child was trapped and submerged under the vehicle for approximately 15 minutes. He was pulled to land by his mother who initiated mouth-to-mouth ventilation. Paramedics arrived several minutes later and found the child asystolic. They began external cardiac compressions, intubated the patient's trachea, and transported the child to the emergency department of the local hospital.

Upon arrival in the local emergency department, the child remained asystolic with a rectal temperature less than 25° C (the lower limit of the thermometer). External chest compressions were continued. Chemical resuscitation with epinephrine and atropine resulted in the restoration of normal sinus rhythm after the child's temperature had risen to 28° C. The total duration of asystole was approximately 40 minutes. The patient was then transported to our tertiary referral center for further management.

Upon arrival in our emergency department, the patient's core temperature was 32° C with a Glasgow Coma Scale* (GCS) score of 4. Initial arterial blood-gas values while being manually ventilated with F_{IO_2} of 1.0 revealed: pH 6.83, P_{aCO_2} 65 torr (8.7 kPa), P_{aO_2} 133 torr (17.7 kPa), and base deficit 24 mmol/L (corrected to a temperature of 37° C). Despite ven-

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* See the Glossary following the References Section.

tilatory support and sodium bicarbonate administration, cardiorespiratory dysfunction progressed. Chest radiograph demonstrated diffuse alveolar disease (Fig. 1). A computerized tomography scan of the patient's head revealed no evidence for anoxic brain injury or cerebral edema. The patient was transferred to the pediatric intensive care unit (PICU).

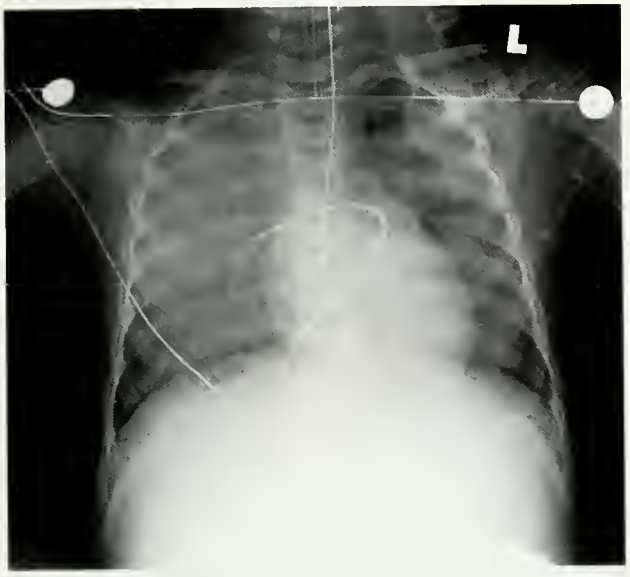

Fig. 1. Chest radiograph immediately after PICU admission. Note the diffuse air space involvement, which is consistent with a diagnosis of acute respiratory distress syndrome.

At the time of PICU admission, the rectal temperature was 36.3° C. The child had a sinus tachycardia at a rate of 170 beats/min and a blood pressure of 110/70 mm Hg. Auscultation of the lungs revealed diffuse coarse rales and rhonchi with decreased breath sounds. Neurologic examination revealed purposeful movements, appropriate responses to pain, eye opening to pain, and a GCS score of 8.

The patient was mechanically ventilated (Servo 900C, Siemens Medical Systems, Solno, Sweden) in a time-cycled, pressure-limited, decelerating-flow (pressure-control) mode through a cuffed 5.0-mm ID endotracheal tube. Initial ventilator settings included F_{IO_2} 0.50, peak inspiratory pressure (PIP) 38 cm H_2O, positive end-expiratory pressure (PEEP) 10 cm H_2O, inspiratory time (t_I) 1.0 second, and respiratory rate 20 breaths/min. These settings resulted in arterial blood-gas values of pH 7.47, P_{aCO_2} 31 torr (4.1 kPa), P_{aO_2} 94 torr (12.5 kPa), and base deficit 0 mmol/L. A balloon-tipped, flow-directed catheter was positioned in the right pulmonary artery enabling hemodynamic measurements. Cardiac index by the thermodilution technique was 5.8 $L \cdot min^{-1} \cdot m^{-2}$, pulmonary artery pressure 21/10 mm Hg, central venous pressure 15 mm Hg, and pulmonary artery wedge pressure 12 mm Hg.

During the first 24 hours in the PICU, the child developed severe combined cardiorespiratory dysfunction. His P_{aO_2} decreased to 42 torr (5.6 kPa) with an oxyhemoglobin saturation (S_{aO_2}) of 71% despite our having increased the F_{IO_2} to 1.0, PIP to 40 cm H_2O, PEEP to 14 cm H_2O, and $\bar{P}_{aw}$ to 25 cm H_2O. The t_I was maintained at 1.0 second, and the respiratory rate at 20 breaths/min. Ventilation remained adequate with the P_{aCO_2} ranging from 35 to 45 torr (4.7 to 6.0 kPa). A trial of high-frequency oscillatory ventilation was ineffective with oxygen saturation decreasing to 60-65% despite an increase in $\bar{P}_{aw}$ to 35-40 cm H_2O. Large amounts of frothy, red secretions were suctioned hourly from his endotracheal tube.

Cardiovascular instability developed with recurrent episodes of severe hypotension (systolic blood pressure less than 40 mm Hg), bradycardia (pulse less than 80 beats/min), and ventricular ectopy. An electrocardiogram revealed ST-segment depression in all leads, while an echocardiogram demonstrated a hypocontractile left ventricle with minimal motion of the posterior free wall. Creatinine kinase MB fraction was elevated at 36 IU/L (normal < 9 IU/L). Despite continuous inotropic support with epinephrine 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$, dopamine 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and dobutamine 15 $\mu g \cdot kg^{-1} \cdot min^{-1}$, cardiovascular function continued to deteriorate. The cardiac index decreased to 2.2 $L \cdot min^{-1} \cdot m^{-2}$ while the pulmonary artery pressure increased to 54/41 mm Hg, the central venous pressure increased to 27 mm Hg, and the pulmonary artery wedge pressure increased to 33 mm Hg. The serum lactate rose from 1.9 to 6.0 mmol/L indicating inadequate systemic tissue oxygen delivery and the onset of anaerobic metabolism. Resuscitative efforts included multiple boluses of epinephrine, lidocaine, calcium, and colloid. Throughout this period of cardiopulmonary instability, the results of the child's neurologic examination remained unchanged.

Approximately 30 hours after the near-drowning accident, the decision to initiate ECMO for refractory cardiorespiratory dysfunction was made. The patient was placed on veno-arterial ECMO via the right common carotid artery and the right internal jugular vein (Fig. 2) utilizing ECMO flow rates of 100 mL/kg. The ventilator was changed to time-cycled, continuous-flow, volume-limited mode with variable flow waveform and pressure support. Ventilator settings included respiratory rate 8 breaths/min, tidal volume 120 mL, PEEP 10 cm H_2O, pressure support 5 cm H_2O, and F_{IO_2} 0.21. The patient's P_{aO_2} ranged from 80 to 120 torr (10.7 to 16 kPa). Activated clotting times were maintained between 160 and 175 seconds. The chest radiograph is shown in Figure 3. Pharmacologic paralysis was discontinued, and the child showed a progressive increase in spontaneous movements and eye opening. Serial electroencephalographic examinations revealed no concerning abnormalities.

After the initiation of ECMO, the heart developed cardiac stun, which is the near-total or total absence of left ventricular contribution to cardiac output.[14] This continued for approximately 36 hours. Repeat echocardiogram on ECMO Day 3

revealed globally depressed myocardial function, but no focal wall-motion abnormalities. During these several days, the electrocardiogram became normal, and the chest radiograph showed progressive improvement as PEEP was maintained at 10 cm H_2O.

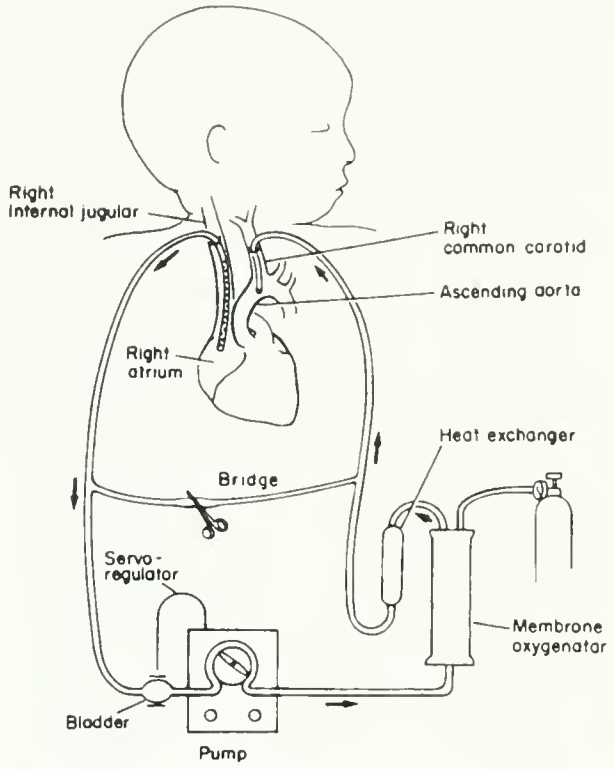

Fig. 2. Diagram showing the components of and the direction of blood flow through a venoarterial extracorporeal membrane oxygenator circuit.[13]

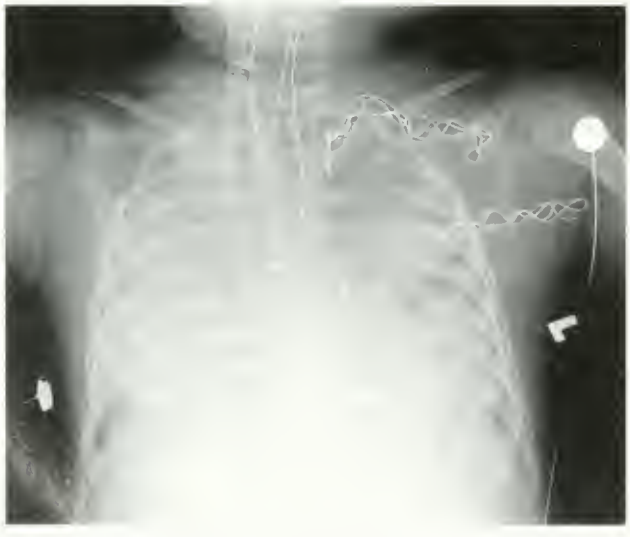

Fig. 3. Chest radiograph after the initiation of ECMO shows a worsening of atelectasis—a common occurrence as ventilator settings are reduced in a lung-sparing strategy.

ECMO was successfully discontinued after an uncomplicated 137-hour course. The child remained hemodynamically stable off ECMO with no vasopressor requirements. Ventilator settings in the pressure-control mode included respiratory rate 16 breaths/min, PIP 40 cm H_2O, PEEP 8 cm H_2O, and F_{IO_2} 0.60. P_{aO_2} ranged from 80 to 100 torr (10.7 to 13.3 kPa).

Six days after vessel decannulation, the patient's trachea was successfully extubated (hospital Day 13). At that time, he was awake and communicating with his family and the staff. Two days later, the patient was transferred to an inpatient ward. One month after his near-drowning incident, the child was discharged home in excellent condition. Three months after discharge, the child was determined to have normal cardiac, pulmonary, and neurologic function. Follow-up studies, including electrocardiogram, chest radiograph (Fig. 4), crying vital capacity, end-tidal capnography, room-air oximetry, and magnetic resonance imaging of his head, were normal.

Fig. 4. Chest radiograph 3 months after discharge from the hospital demonstrating resolution of the ARDS.

Discussion

Controversy exists regarding the prognosis of submersion victims. The major factors that separate intact survivors from impaired survivors and nonsurvivors have been extensively studied; however, the results are often conflicting.[6,15,16] Thus, the use of 'heroic measures' in such a patient remains controversial. Whether an asystolic submersion victim should be resuscitated is dilemmatic and how best to support the cardiorespiratory system in these patients is controversial. Dra-

matic case reports of survival after prolonged cold-water submersions have been reported.[17-23] In several of these cases, cardiopulmonary bypass was used for extracorporeal blood warming, with the longest reported time on cardiopulmonary bypass being just over two hours.[17,22,23] However, prolonged cardiopulmonary bypass in the form of ECMO to support both pulmonary and cardiac function may be a beneficial approach for these patients.

The pulmonary dysfunction data by Levin et al[4] and Biggart and Bohn[6] suggest that pulmonary failure is likely after a submersion accident; however, significant cardiac dysfunction is much less likely to occur.[4,10,11] In our patient, both pulmonary and cardiac failure occurred—a combination that carries a high risk of mortality. The need for near-complete respiratory and cardiac support prompted the decision to initiate venoarterial ECMO.

The acute lung injury that developed in this patient is due to a variety of causes. In addition to the primary lung injury, strong evidence exists that mechanical ventilation with high PIP and F_{IO_2} may cause the initial injury to progress. The ventilatory management of this patient prior to ECMO focused on minimizing PIPs and utilizing pressure-limited ventilation and a decelerating flow pattern (pressure-control). PEEP was titrated to re-establish lung volume (functional residual capacity, FRC) while the F_{IO_2} was decreased when possible. Normocapnia (P_{aCO_2} 35-45 torr) was used to preserve arterial pH in the face of the metabolic acidosis caused by cardiac failure and poor systemic perfusion. Permissive hypercapnia and inverse ratio ventilation were not instituted due to the metabolic acidosis, progressive pulmonary artery hypertension, and decreasing cardiac indices. Our inability to adequately oxygenate this patient coupled with the adverse effects of maximal ventilatory support and cardiac failure contributed to the decision to initiate ECMO.

During ECMO, the ventilator settings were markedly reduced; however, PEEP levels of 10-12 cm H_2O were used to maintain end-expiratory lung volume—FRC. Spontaneous ventilation and patient-ventilator synchrony were facilitated by pressure-support ventilation. Lung recovery was monitored with daily measures of dynamic and static compliance and with chest radiographs. As compliance and cardiac output improved, ventilatory support was increased and ECMO support decreased.

One rare complication that occurred in this patient was the development of cardiac stun. Cardiac stun has been reported to occur in approximately 5% of infants on venoarterial ECMO;[24] however, its incidence in older patients is not known. This phenomenon is thought to occur secondary to reperfusion of the heart, an increased cardiac afterload from the high ECMO flow rates, a preferential coronary blood supply from the poorly oxygenated pulmonary venous return, and/or an increase in the left-ventricular end-diastolic pressure. An elevated left ventricular end-diastolic pressure may occur if the left ventricle is unable to empty and ventricular pressure increases. If this increase in left ventricular end-diastolic pressure is excessive, it may adversely affect both pulmonary and cardiac function. Increases in left ventricular end-diastolic pressure cause elevations in left atrial pressure with secondary pulmonary venous hypertension, pulmonary edema, and delayed pulmonary recovery. In addition, coronary perfusion pressure and coronary blood flow depend on the difference between the left ventricular end-diastolic pressure and the aortic diastolic pressure. Therefore, an increase in left ventricular end-diastolic pressure results in a decrease in coronary perfusion pressure, coronary blood flow, and myocardial oxygen delivery. This may delay restoration of normal myocardial function.

Cardiac stun is, therefore, a potentially dangerous condition with negative effects on both the cardiac and pulmonary systems. If cardiac stun occurs, 2-dimensional echocardiography may be helpful in following its resolution, which usually occurs within 72 hours.[24] However, if spontaneous resolution does not occur, decompression of the left ventricle may be attempted by either atrial septotomy or direct transthoracic cannulation of the left atrium. This latter option involves diverting blood from the left atrium to the venous side of the ECMO circuit, decreasing left ventricular end-diastolic pressure and improving cardiac and pulmonary function.

Recovery can be achieved for both cardiac and pulmonary failure in the near-drowning patient. The primary difficulty lies in identifying the submersion victim with cardiopulmonary failure who is neurologically intact and, therefore, a potential ECMO candidate. Major prognostic variables are the temperature of the water and of the patient. There is strong evidence that rapid hypothermia, core temperature less than 32° C, significantly improves the prognosis for neurologically intact survival after a submersion accident.[6,17-21,25-27]

Several studies have researched neurologic outcome based on the GCS score. A GCS score of ≤ 5 at the time of PICU admission has been correlated with a poor outcome.[15,16] A GCS score of ≥ 6 has been related to intact survival.[28] Whether hypothermia modifies these findings is unclear. GCS score determinations after warming may be more prognostic than the initial GCS score. As demonstrated in our patient, the GCS score increased from 4 in the emergency department to 8 in the PICU with warming and routine resuscitative care. Therefore, our patient would have been predicted to have a successful neurologic outcome based on the GCS taken after warming.

In Summary

When the neurologic status of such a patient is encouraging, ECMO may be lifesaving if pulmonary and/or cardiac failure is occurring. This report illustrates the successful use of ECMO in the case of a young victim of cold-water-submersion in whom neurologic function was preserved, but both pulmonary and cardiac function were severely impaired. It is often difficult to assess the appropriateness of ECMO for the treat-

ment of a submersion victim. Taking this case and the published data into consideration, we offer the following suggestions concerning the appropriateness of ECMO in victims of near-drowning.

- The patient should first meet the ECMO criteria of the individual institution.
- A patient should not be excluded as an ECMO candidate based upon the total duration of asystole.
- The temperature of the water and of the patient must be taken into consideration.
- If central nervous system pathology is present, including hemorrhages, cerebral edema, and anoxic brain injury, ECMO should not be considered.

ECMO may be lifesaving for victims of submersion accidents who are neurologically intact but have life-threatening cardiac and/or pulmonary failure. If a patient meets ECMO criteria from a cardiopulmonary status, has no obvious central nervous system pathology, and has an encouraging neurologic examination, a trial of ECMO may be indicated.

If, after the initiation of ECMO, the patient's neurologic status deteriorates or fails to improve appropriately, early withdrawal of ECMO should be considered.

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Glossary for ECMO & Cold-Water Submersion

afterload: the 'downstream' resistance imposed on each of the ventricles. The afterload of the right ventricle is the force opposing ejection caused by the resistance of the pulmonary vasculature; whereas, the afterload of the left ventricle is the force opposing ejection caused by the resistance of the systemic circulation. Reducing afterload of an impaired ventricle often improves its function.

atrial septotomy: the intentional creation of an opening in the atrial septum, either to create atrial level mixing for patients with transposition of the great vessels or to provide an alternate path for systemic blood flow in the case of severe right ventricular dysfunction. In this latter case, systemic perfusion is maintained at the expense of a lower arterial oxyhemoglobin saturation. Atrial septotomy may also be an option to relieve end-diastolic engorgement of the left ventricle, such as that seen in the case we report.

cardiac index: the **cardiac output** divided by body surface area. Cardiac index ($L \cdot min^{-1} \cdot m^{-2}$) enables the comparison of values of **cardiac output** among patients of varying size and to the 'normal' values that have been developed.

cardiac stun: the near-total or total absence of left ventricular contribution to **cardiac output** after the initiation of **venoarterial ECMO**, characterized by increased left-ventricular **afterload** and increased end-diastolic volume.

cardiac output: the volume of blood ejected by the heart during a given time period, usually L/min. Cardiac output is the product of stroke volume and heart rate.

central venous pressure: Central venous pressure is obtained from a transduced central venous catheter placed in an internal jugular or subclavian vein with the catheter tip in the superior vena cava. The central venous pressure aids in assessing the intravascular volume status and, indirectly, right ventricular function.

colloid infusions: the intravenous administration of colloids such as plasma protein (including albumin) and blood products. Routine intravenous fluids, such as D_5 0.45% saline, are termed crystalloids.

coronary perfusion pressure: Coronary perfusion pressure determines coronary blood flow and depends on the difference between the left ventricular end-diastolic pressure and the aortic diastolic pressure. Therefore, an increase in left-ventricular end-diastolic pressure results in a decrease in coronary perfusion pressure, coronary blood flow, and myocardial oxygen delivery.

crying vital capacity: Vital capacity is the volume of gas that can be maximally expired following a maximal inspiration to total lung capacity. Crying vital capacity is a test of vital capacity in children who cannot cooperate for even a minimal voluntary vital capacity effort. Crying is induced in the infant, and the presumption is that a reflex inspiration to total lung capacity occurs, which can be measured.

2-D echocardiography: the use of ultrasonic waves to create an image of the heart and central vessels. Such images are obtained by steering the transducer crystal to create a pie-wedge-shaped image of the heart. Various portions of the heart are then viewed relative to each other in that plane, providing precise anatomic definition.

ectopy: an inappropriately timed, initiated, or conducted heart contraction or series of contractions, which may be initiated in the atria or ventricles.

extracorporeal membrane oxygenation (ECMO): ECMO is life-support technology designed to provide near-total respiratory gas exchange and circulation. ECMO may be either venoarterial or venovenous. In both forms of ECMO, blood is removed from the patient, oxygen and carbon dioxide exchanged across an artificial membrane. The blood is then warmed and then returned to the patient.

Glasgow Coma Scale (GCS) score: assigns scores for clinical findings that can develop following head trauma and/or brain injury. The assessment categories include eye opening, motor response, and verbal response to different degrees of stimulation. The scale ranges from 3 (no neurologic responses) to 15 (normal responses). GCS scores have been extensively correlated with survival and impairment.

left-ventricular end-diastolic pressure: the pressure measured in the left ventricle during diastole. In the absence of mitral-valve disease, this value is approximated by the **pulmonary artery wedge pressure**.

pulmonary artery wedge pressure: This pressure is obtained from a balloon-tipped pulmonary artery catheter. Inflation of the balloon causes migration of the tip until the balloon wedges into a smaller arterial branch. The pressure measured is an estimate of the left atrial pressure.

ST-segment depression: Depression of the ST-segment on an electrocardiogram is an indication of myocardial ischemia.

venoarterial ECMO: refers to an ECMO circuit by which blood is removed from the right atrium via a cannula in the internal jugular vein and is returned into the aortic arch via a cannula placed in the carotid artery.

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Textbook of Bronchoscopy, edited by SH Feinsilver and AM Fein. Hardcover, illustrated, 539 pages. Baltimore: Williams and Wilkins, 1995. $125.

The **Textbook of Bronchoscopy** joins a recently expanding field of bronchoscopy books that span a range of purposes. On the one hand, readers interested in bronchoscopy can now find relatively short atlases of normal and abnormal bronchoscopic findings and, on the other, exhaustive texts about the technique of bronchoscopy, related equipment, and the diagnostic value of bronchoscopy. Within this spectrum of books, **Textbook of Bronchoscopy** contributes a comprehensive text that addresses practical aspects of bronchoscopy such as techniques with the flexible fiberoptic bronchoscope (eg, bronchoalveolar lavage, transbronchial needle aspiration, and transbronchial biopsy); diseases for which bronchoscopy is frequently performed; and complementary or alternative procedures including rigid bronchoscopy, medical thoracoscopy, and nonbronchoscopic lung lavage.

Edited by two recognized authorities on bronchoscopy, the book is multi-authored with contributions from many highly regarded experts. The book is organized into 4 sections that include 33 separate chapters written by 59 contributors. These sections are:

- Technical aspects of fiberoptic bronchoscopy (consisting of 12 chapters addressing indications and contraindications for bronchoscopy, preparing patients for flexible fiberoptic bronchoscopy, equipment and its handling, and aspects of training).
- Diagnostic problems (consisting of 10 chapters reviewing specific diseases for which bronchoscopy is commonly performed and issues specifically related to the role of bronchoscopy in diagnosis and management).
- Therapeutic bronchoscopy (consisting of 4 chapters that discuss therapeutic applications, including foreign body removal, laser, stent placement, and endobronchial irradiation).
- Related techniques (consisting of 7 chapters reviewing other chest procedures, two of which are occasional alternatives to flexible fiberoptic bronchoscopy [nonbronchoscopic lavage and rigid bronchoscopy]).

Overall, the organization of the book is logical and the chapters are complete, authoritatively referenced, and well-illustrated. For example, the chapter "Care and Maintenance of the Flexible Bronchoscopy" by Drs Lee and Mehta provides photographs depicting a number of damaged bronchoscopes that nicely instruct about the hazards of mishandling these instruments. The chapter "Hemoptysis" by Drs Lippman and Higgins includes a number of helpful tables, with several that present exhaustive summaries of relevant literature. Also, the chapter entitled "Atlas and Basic Technique" by Drs Kitamura and Kobayashi contains 8 full-page color plates (with 32 individual photographs) depicting normal endobronchial anatomy and 25 additional black-and-white photographs depicting abnormal endobronchial findings such as various neoplasms, granulomatous and infiltrative airway lesions, and tracheopathia osteoplastica. This reader's preference to see all color photographs must be tempered by the realities of expense and price and the editors' purpose to "go beyond several of the available atlases of bronchoscopy to produce a comprehensive text encompassing all aspects of procedure." This book clearly succeeds in this goal and is an authoritative text about bronchoscopy.

As discussed by Dr Bone in the foreword and the editors in the preface, the intended target audience for this book is "students and practitioners of chest medicine." While this audience certainly includes respiratory care practitioners, RCPs involved with flexible fiberoptic bronchoscopy are likely to have the greatest interest in the first 2 sections of the book ("Technical Aspects" and "Diagnostic Problems") and to seek information in sections 3 ("Therapeutic Bronchoscopy") and 4 ("Related Techniques") for occasional reference.

Overall, **Textbook of Bronchoscopy** offers a comprehensive review of flexible fiberoptic bronchoscopy and related bronchoscopic techniques that will be a valuable resource for every bronchoscopy suite. Respiratory care practitioners who practice in an intensive care unit or who assist with bronchoscopy will find this book offers much-needed information and much more.

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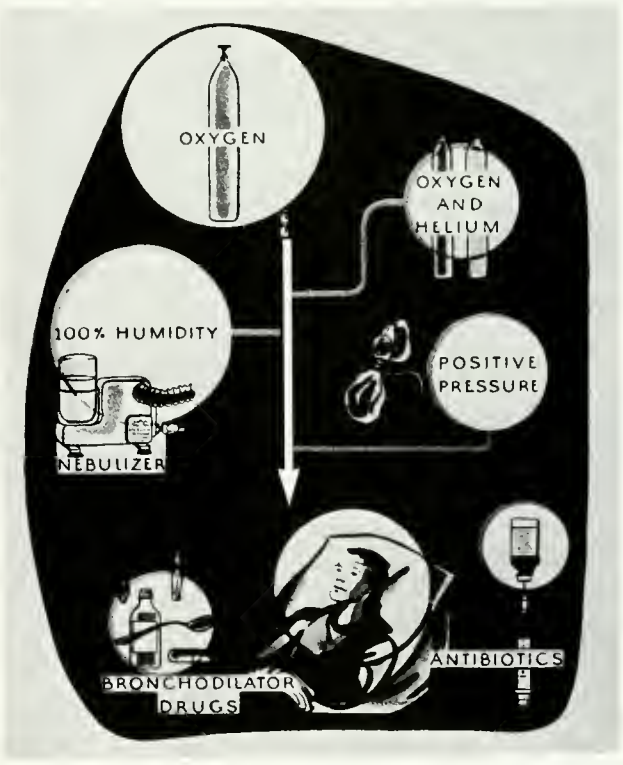
Effective Inhalation Therapy: Generalized Obstruction

Generalized obstruction is always incomplete, since complete obstruction of all the bronchi is not found in the living patient. In such generalized disease there is more interference in the small bronchi than in the major bronchi and trachea. This is because the causative factor is a swelling or inflammation of the bronchial mucosa, accompanied by more or less mucus secretion which is generally rather tenacious. Since the difference in size of the bronchi is much greater than the difference in the amount of secretion or swelling of the lining, it is clear that the same amount of interference with the lumen may cause complete obstruction in the bronchiole and very little obstruction in the major bronchus. Furthermore, because of the mechanism of narrowing and shortening of the bronchus on expiration, the obstruction is greatest in the expiratory phase.

Generalized obstruction is most commonly found in asthma, tracheo-bronchitis, or traumatic bronchitis associated with the inhalation of smoke or irritating gases. In all of these conditions the inflammatory reaction will vary with the degree of severity of the disease. The obstruction thus produced may cause changes which will vary from a wheezing on expiration to almost complete interference of the passage of air. The symptoms are primarily those of interference with ventilation. The earliest and greatest interference is in the expiratory phase for the reasons mentioned above. Consequently, the earliest symptom is an expiratory wheeze. This is soon followed by a rapid shallow respiration, dyspnea, difficulty on inspiration and all the symptoms of acute asphyxia.

Although it is necessary to treat the underlying condition if one is to eliminate the bronchial obstruction, it is just as necessary to treat the difficulty in ventilation and the anoxia. For this, inhalation of oxygen in percentages of 50 per cent and higher should be used. If the obstruction is not too severe, this increase in percentage will raise the per cent of alveolar oxygen to give adequate saturation of the hemoglobin. This will cause a slower and easier respiration and frequently by this means alone alleviate a great deal of the obstruction of the small bronchi.

However, if the obstruction is more severe as evidenced by much wheezing and dyspnea, it is advisable to take advantage of the benefits of positive pressure breathing and frequently of helium and oxygen combinations. Positive pressure on expiration, accomplished by the use of the positive pressure mask, will act to prevent the complete closure of the small bronchi and thus maintain the airway for the following inspiration. By this means and also by the reason of



Treatment of generalized incomplete bronchial obstruction is directed at freeing the airway, removing possible infection, and maintaining an adequate oxygen level in the alveoli while this is taking place.

From Effective Inhalation Therapy, written by Edwin R Levine MD in cooperation with Alvan L Barach MD, J Winthrop Peabody MD, and Maurice Segal MD, and published in 1953 by the National Cylinder Gas Co of Chicago. Dr Levine, an anesthesiologist, was an early champion of our profession and President of the American Association for Inhalation Therapy in 1952 (the only physician in the Association's history to so serve). The other authors were also pioneers in pulmonary medicine and inhalation therapy.

This excerpt was made available to RESPIRATORY CARE by Teri Nikolai Wilson RRT RPFT, The Children's Medical Center, Dayton, Ohio.

increased intra-alveolar pressure, the mechanism producing pulmonary edema is reversed, and thus may be eliminated. The procedure for proper use of the positive pressure mask should be followed with great care if the proper result is to be obtained. Where there is a great deal of difficulty in inspiration, some help may be given by the use of apparatuses which provide positive pressure inspiration. This will decrease the amount of work necessary to cause air to enter the markedly diminished spaces which lead to the alveoli.

In any case where the obstruction is marked and the discomfort of the patient very great, one should not use the above methods alone for very long unless a definite improvement is noticed. If such improvement does not occur rapidly or if the severity of symptoms is sufficient, helium and oxygen mixtures should be used instead of oxygen alone. This mixture by reason of its lighter mass will be enabled to pass through the narrowed spaces and carry a larger volume of gas into the alveoli than is possible with air or oxygen alone. However, these patients are suffering from severe oxygen deficiencies. Therefore, as high a concentration of oxygen as is feasible under the circumstances should be used. This should never be less than 30 per cent and in many cases 40 to 50 per cent of oxygen, with the remainder of the gas helium. Again it is necessary to emphasize that helium and oxygen mixtures can only he given with a properly fitting mask or hood and should not be used in adults with any other type of apparatus.

Because infection is so often present in generalized bronchial obstruction, it is not incorrect to say that the inhalation of aerosol penicillin or other antibiotics, using adequate nebulizing equipment, should be a regular part of the treatment. In a great many cases the use of broncho-dilator drugs by this means or by other modes of administration is of tremendous value.

Here as in all generalized bronchial irritations it is of paramount importance, indeed in some cases actually a life saving matter, that the inspired gas be properly humidified. It is desirable that this gas have not less than 100 per cent relative humidity. This can only be obtained with a proper humidifying apparatus, and the nebulizer type is recommended. In fact with a proper large volume nebulizer, oxygen, antibiotics, broncho dilators, and water vapor can be introduced simultaneously.

These conditions, with the occasional exception of bronchial asthma, improve slowly. The improvement of the patient's symptoms may be much more rapid than the actual change in the pathology. It is a common mistake to stop inhalation therapy too early in the belief that the patient is getting an adequate amount of oxygen by his unassisted respiration. It is much better to continue this therapy with a gradually tapering off process when the pathology is entirely or almost entirely cleared up. A common fallacy is that if a patient receives oxygen his breathing will be more shallow and that therefore bronchial obstruction and atelectasis may occur more easily. This is not so. The dyspneic patient has a tendency to breathe rapidly but the respirations are shallow. This is particularly true in generalized bronchial obstruction, since the effort of breathing causes bronchial spasm and interferes with ventilation. The relaxed type of breathing that is associated with oxygen therapy will not only furnish adequate oxygen and protect the patient from fatigue but, because of its ease, tends to maintain an adequate airway and guarantees more uniform aeration of the lung.

Understand.
Adapt.
Prosper!

AARC

42nd International Convention & Exhibition
November 3-6 • San Diego, California

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page ____ of

Form Approved OMB No 0910-0291 Expires 1/31/96
See OMB statement on reverse

FDA Use Only (RESP CARE)

Triage unit sequence #

A. Patient information

1. Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4. Weight
____ lbs
or
____ kgs

B. Adverse event or product problem

1 ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2 Outcomes attributed to adverse event (check all that apply)
☐ death ____ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other:

3 Date of event (mo day yr)

4 Date of this report (mo day yr)

5 Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4 Diagnosis for use (indication)
#1
#2

5 Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7 Exp. date (if known)
#1
#2

8 Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only)
– –

10 Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2. Type of device

3 Manufacturer name & address

4 Operator of device
☐ health professional
☐ lay user/patient
☐ other:

5. Expiration date (mo/day/yr)

6.
model #
catalog #
serial #
lot #
other #

7 If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10 Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional?
☐ yes ☐ no

3 Occupation

4 Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5 If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 (6/93)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send your comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

Reports Clearance Officer, PHS
Hubert H. Humphrey Building,
Room 721-B
200 Independence Avenue, S.W.
Washington, DC 20201
ATTN: PRA

and to:
Office of Management and Budget
Paperwork Reduction Project
(0910-0291)
Washington, DC 20503

Please do NOT return this form to either of these addresses.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service • Food and Drug Administration

FDA Form 3500-back

Please Use Address Provided Below – Just Fold In Thirds, Tape and Mail

Department of Health and Human Services
Public Health Service
Food and Drug Administration
Rockville, MD 20857

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Penalty for Private Use $300

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MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787

1996 Summer Forum
July 12–14
Registry Hotel, Naples, FL

Sail Away With Us To
San Diego
American Association for Respiratory Care 42nd Annual Convention and Exhibition
Nov. 3–6, 1996, Sunday–Wednesday, San Diego, CA

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

The Lambda Beta Society

The National Honor Society for the Profession of Respiratory Care

For information about the Lambda Beta Society—the National Honor Society for the Profession of Respiratory Care, contact the Society Office at 1701 W Euless Blvd, Suite 200, Euless TX 76040, (817)283-4269.

Call for Abstracts
1996 RESPIRATORY CARE OPEN FORUM

Deadline: April 28, 1996

Accepted abstracts will be printed in the October 1996 issue of RESPIRATORY CARE

Selected authors will present their research at the OPEN FORUM during the AARC 42nd International Convention & Exhibition in San Diego, California November 3-6, 1996

The National Board for Respiratory Care

8310 Niemann Road
Lenexa KS 66214

For information about
1996 Examination & Fee Schedule

call (913) 599-4200
or FAX (913) 541-0156

Vincent Kracum, 68, died of pneumonia February 3, 1996 at Scottsdale Memorial Hospital in Scottsdale, Arizona. Kracum began his career in respiratory therapy in 1946 as a Navy technician in "oxygen therapy." Throughout his life he contributed to the profession by playing a part in the formation of 72 hospital departments for respiratory care in the U.S. and Canada and by serving on the National Board for Respiratory Care, the American Association for Respiratory Care's Nominating Committee and Board. He was awarded the Greater New York Affiliate's Golden Tree of Life Award in 1973. Memorials may be made to the Arizona Lung Association, the American Heart Association, or the American Respiratory Care Foundation.

Bill E Morrison, 61, who became registered Respiratory Therapist Number 60 in 1962 and who served the profession, the American Association for Respiratory Care, and the American Respiratory Care Foundation in many ways, died February 12, 1996 at his home in Lebanon, Missouri. Memorial contributions may be made to the American Respiratory Care Foundation, 11030 Ables Lane, Dallas TX 75229.

Ernst Trier Mörch, an inventor, anesthesiologist, and a member of the Danish resistance during World War II, died January 13, 1996 in New York. He was 87 years old.

While in Denmark during the German occupation, Mörch used a sewer pipe attached to a piston and motor to invent the Mörch Piston Respirator—the forerunner of modern ventilators. Senior respiratory care practitioners may be familiar with the Mörch I Ventilator, Mörch II 'Surgical' Ventilator, or the Mörch III 'Piston' Ventilator. His inventiveness was further used to aid in the Danish resistance, credited for saving all but 500 of the country's 7,000 Jews. When the Gestapo used bloodhounds to sniff out hiding Jews, Mörch and a pharmacist mixed dried rabbit's blood and cocaine to effectively detour the dogs.

In 1949, Mörch came to the United States working first in Washington and then Chicago. (Source: *Washington Post*, January 20, 1996.) (Courtesy: Harry Kirshenbaum MEd RRT, El Paso, Texas.)

1996 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Convention & Exhibition in San Diego, California, November 3-6, 1996. Accepted abstracts will be published in the October 1996 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method or device, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method/device evaluation. Abstract *must* include (1) Background: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: Relevant basic information important to understanding the case. (2) Case Summary: Patient data and response, details of interventions. (3) Discussion: Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form, *and simple figures may be included provided they fit within the space allotted.* No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested at the bottom of abstract form. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts postmarked by February 11, 1996 will be reviewed and the authors notified by letter only to be mailed by March 22, 1996. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the final deadline (April 28, 1996).

Final Deadline

The mandatory Final Deadline is April 28 (postmark). Authors will be notified of acceptance or rejection *by letter only.* These letters will be mailed by July 15, 1996.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

1996 Respiratory Care Open Forum

Abstract Form

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, leave a 'ragged' right margin).
4. **Do not use type size less than 10 points.**
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original & 1 photocopy (along with postage-paid postcard) to

Respiratory Care Open Forum
11030 Ables Lane
Dallas TX 75229-4593

Early deadline is February 11, 1996 (postmark)

Final deadline is April 28, 1996 (postmark)

13.9 cm or 5.5"

Presenter

Name & Credentials

Mailing Address

Voice Phone & Fax

Corresponding Author If Different from Presenter

Name & Credentials

Mailing Address

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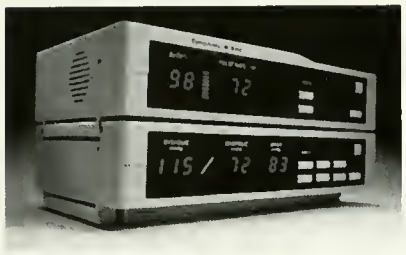
News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229-4593.

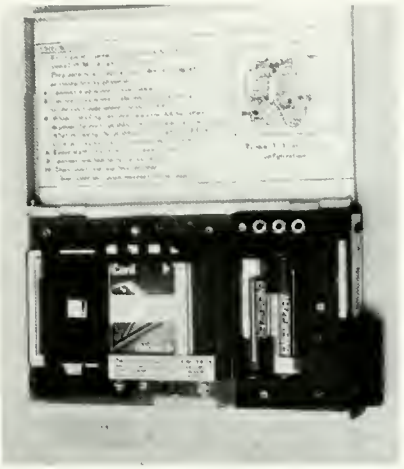
New Products & Services



MONITORING SYSTEM. Nellcor Puritan Bennett releases the Nellcor Symphony™ Monitoring System. According to the manufacturer, the monitoring system is cost effective, flexible, and easy to use because it is a modular system that lets practitioners 'mix and match' monitors according to clinical needs and budget constraints. The first components of the system are the N-3000 pulse oximeter and the N-3100 noninvasive blood pressure monitor—both serve as compact stand-alone monitors and work together for a combination of S_{pO_2} and NIBP monitoring. Communication links between the 2 monitors prevents a nuisance alarm. To learn more about the system, contact Nellcor Puritan Bennett, Dept RC, 4280 Hacienda Drive, Pleasanton CA 94588, or call (800) NELLCOR. Please mention RESPIRATORY CARE.

HAND-HELD SPIROMETER. Micro Direct Inc releases the new MicroLoop II hand-held spirometer. The spirometer's many features include real time, high-resolution graphic display, flow-volume loops, pre- and postbronchodilator comparisons, full interpretation, lung age estimation, and a 200 patient-test memory. The MicroLoop II, according to the manufacturer, can be used as a stand alone spirometer, downloaded to a PC for storage and printouts, or used as a PC-based spirometer with the optional MediBase™ software. For details, call (800) 588-3381 and mention RESPIRATORY CARE.

CONTINUOUS GAS DETECTOR. CEA Instruments Inc introduces the newly improved MD-16, a compact, continuous single gas detector. According to CEA Instruments, the MD-16 is a continuous detector for toxic gases and oxygen. Single units are available for monitoring carbon monoxide, chlorine, hydrogen, hydrogen chloride, hydrogen cyanide, hydrogen sulfide, nitric oxide, nitrogen dioxide, oxygen, or sulfur dioxide gases. The unit mounts to the wall and displays all normal information in a green LED. In addition, the unit's warning alarm is illuminated in yellow; the danger alarm is illuminated in flashing red LED and is accompanied by an audible alarm. The alarm can be tested, silenced, and reset using a remote switch. For more information, write to CEA Instruments Inc, Dept RC, 16 Chestnut Street, Emerson NJ 07630, or call (201) 967-5660. Don't forget to mention RESPIRATORY CARE.



AMBULATORY ECG RECORDER. Brentwood Medical Products offers a new addition to ambulatory ECG recorders, the Brentwood E-Z Scan Digital Recorder. The solid-state device is designed for 24-hour continuous Holter recording, claims the manufacturer. In addition, the recorder has a nonvolatile flash memory card that provides reliability and data integrity, an event button, and an LCD clock. The E-Z Scan Recorder also supports 3-channel patient receptacles with DIN style plugs. Patient data are loaded into a computer via an industry standard PCMCIA slot. Call Brentwood at (800) 624-8950, for more details. Don't forget to mention RESPIRATORY CARE.

SPIROMETER CATALOG. VacuMetrics Inc's VacuMed Division has a free 1996 spirometer catalog that contains descriptions, photographs and prices for 7 different spirometers. According to the company, the catalog includes a useful cross-reference chart for the comparison of each spirometer's features. For a copy of the catalog, contact VacuMed, Dept RC, 4483 McGrath Street #102, Ventura CA 93003, (800) 235-3333. Please mention RESPIRATORY CARE when you call.

RESPIRATORY CARE

Manuscript Preparation Guide

General Information

RESPIRATORY CARE welcomes original manuscripts related to respiratory care and prepared according to these Instructions. Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for all aspects of the manuscript and receive galleys to proofread before publication. Each accepted manuscript is copyedited so that its message is clear and it conforms to the Journal's style. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission.

Editorial consultation is available at any stage of planning or writing. On request, specific guidance is provided for all publication categories. These Instructions and related materials are available. Write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593, call (214) 243-2272, or fax (214) 484-6010.

Publication Categories & Structure

Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included. Mark "For publication."

Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

Preparing the Manuscript

Print on one side of white bond paper, 8.5 in. x 11 in. (216 x 279 mm) with margins of at least 1 in. (25 mm) on all sides of the page. Use ***double-spacing*** throughout the ***entire*** manuscript. Use a standard font (eg, Times, Helvetica, or Courier) at least 10 points in size, and do not use italics except for special emphasis. Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. Do ***not*** put authors' names or other identification anywhere except on the title page. Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product-Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

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Article in a journal carrying pagination throughout volume:

1. Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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AARC & AFFILIATES

March 13-14 in Newport, Rhode Island. The RISRC presents its 12th Annual Newport Challenge, "The New Wave of Change," at the Doubletree Hotel. Topics include market-driven changes in health care, therapist-driven protocols, home-care dynamics, opportunities outside acute care settings, and respiratory issues in newborn transport. Participants can earn 11 hours of CRCE credit. Contact Phyllis Shier at (401) 737-2312.

March 13–15 in Southshore Lake Tahoe, California. Chapter 9 of the CSRC presents the 18th Annual Tahoe Conference, "Setting the Sails into the Winds of Change," at the Embassy Suites. CRCE credit has been requested. Call Kathy Gallego, (510) 947-5375 or George Rice, (510) 947-5292.

April 2 via Satellite. The second session of the 7-part, 1996 AARC video conference "Professor's Rounds in Respiratory Care" is "Managing the Ventilator: What and When." The conference covers setting up the ventilator, selecting appropriate settings, monitoring the patient, and making appropriate adjustments. The video conference is from 12:30-2 pm Eastern Time; viewers of the live presentation on April 2 or participants in the live telephone question-and-answer session on April 22 receive 1 CRCE credit hour. To register, call the AARC at (214) 243-2272.

April 2-4 in Lake George, New York. The New York State Society for Respiratory Care hosts its Annual Symposium at the Omni Sagamore Hotel. CRCE credit has been requested. Call Craig Hilligas at (518) 262-3563.

April 3-5 in Gatlinburg, Tennessee. The TSRC presents its annual convention and exhibition, "Another Walk on the Wild Side," at the Park Vista Hotel overlooking the Smoky Mountains. Sessions cover clinical management and student and home care issues. CRCE credit has been requested. Contact Colleen Schabacker at (615) 384-1731.

April 10-12 in Baton Rouge, Louisiana. The LSRC announces its Annual Education Meeting, "Respiratory Gumbo," at the Crown Sterling Suites. Topics include intratracheal ventilation, a new technique for minimal tidal-volume ventilation; quality control; and integrated delivery systems and RC departments. The convention features exhibits, the Crawfish Boil, and the Pelican Bowl competition. CRCE credit has been requested. Call Jim Lanoha at (504) 381-6542.

April 11-12 in Lufkin, Texas. Pineywood's Chapter of the TSRC announces its 8th Annual Seminar. Topics include pressure support/flow-by, lactic acid electrolyte interpretation, and noninvasive ventilation. Contact Sheila Sanchez at (409) 639-7006.

April 17-18 in Salt Lake City, Utah. The USRC announces its Annual Convention at the Utah State Fair Park in the Grand Building. Dr Forrest Bird is the keynote speaker. Sessions cover lung reduction procedures, advanced directives in living wills, a fast-track weaning protocol, and alternate gases for ventilation. Contact Weber State University's Respiratory Care Department at (801) 626-7071.

April 17-19 in Birmingham, Alabama. The ASRC hosts its Annual Respiratory Care Conference—the 1996 Educational Olympics—at the Radisson Hotel. AARC President Charlie Brooks presents the keynote address on the role of the respiratory therapist in managed care. Activities include the ASRC Sputum Bowl, Respiratory Olympics, and exhibits. CRCE credit has been requested. Contact Karla Gray at (334) 705-3790.

April 17-19 in Lawrence, Kansas. The Kansas Respiratory Care Society announces its 19th Annual Education Seminar, "Somewhere Over the Rainbow Is Clinical Care," at the Lawrence Holidome. Topics include pulmonary clearance techniques, asthma, graphic waveform and loop analysis in mechanical ventilation, and volume reduction for emphysema. CRCE credit has been requested. Contact Pat Munzer at (913) 231-1010, extension 1284.

April 18–19 in Palm Springs, California. Chapter 2 of the CSRC presents "Bright Horizons in Respiratory Care," at the Hyatt Regency Suites. Presentations include new modes of mechanical ventilation, invasive pulmonology, and clinical application of metabolic monitoring. CRCE credit has been requested. Contact Tom Taylor at the following phone/fax number: (909) 777-3214.

April 24 -25 in Waterbury, Connecticut. The CSRC presents its annual "Super Symposium" at the Four Points Sheraton. Conference topics include bronchoscopy, weaning protocols, pulmonary rehabilitation, and risk management. Contact Kevin Kenny at (203) 545-3212 or Kathy Glenn-Nadal at (800) 882-0001, extension 42.

April 24-26 in Bismarck, North Dakota. The NDSRC hosts its Annual Educational Symposium, featuring keynote speaker Patrick Reynolds. Topics include ventilator management, pulmonary rehabilitation, cystic fibrosis, and pediatric assessment. Call (701) 224-7870.

April 25-26 in Little Rock, Arkansas. The ASRC and Arkansas Children's Hospital present the Diamond Conference, featuring sessions on pediatric/infant pulmonary functions, partial liquid ventilation, and weaning from mechanical ventilation. Lunch time tutorials include sessions on asthma and therapist-driven protocols. Contact Mike Anders or Kathy Boyle, Respiratory Care Services, Arkansas Children's Hospital, Slot 303, 800 Marshall St, Little Rock AR 72202, (501) 320-3535, fax (501) 320-3411.

April 30-May 3 in Dearborn, Michigan. The MSRC announces its annual meeting, "Crossroads of Opportunity," at the Hyatt Regency. Activities include a Basic Life Support Instructor course and sessions covering assessment, therapist-driven protocols, health care reform, and marketing the RC department. Contact the MSRC office at (517) 336-7605.

May 1-3 in Sioux Falls, South Dakota. The SDSRC hosts its annual meeting at the Howard Johnson. Dr Forrest Bird, the keynote speaker, addresses the role of cardiopulmonary practitioners and airway management. Other activities include an AARC update and lectures on cystic fibrosis, asthma, pulmonary rehabilitation, and surgical alternatives to obstructive lung disease, each preceded by case

study presentations. CRCE credit has been requested. Contact Mary Reinesch at (605) 333-6477.

May 18-21 in Spokane, Washington. The Respiratory Care Society of Washington announces the 23rd Annual Pacific Northwest Regional Respiratory Care Conference at Cavanaugh's Inn at the Park. Sessions cover topics in management, pediatrics, education, pulmonary functions, skilled nursing facilities, and critical care. Healthcare equipment is on display for two days; other activities include a volleyball tournament and a boat cruise. Contact Bob Bonner, Chair of the Health, Education and PE Division, Highline Community College, PO Box 98000, Des Moines WA 98198, (206) 878-3710, extension 3469.

May 21 via Satellite. The third session of the 7-part 1996 AARC video conference "Professor's Rounds in Respiratory Care" is "Managing Demand for Respiratory Care Services." Viewers may learn how to identify the major components of a successful utilization control program and potential demand management roles outside the acute care facility. The conference is 12:30–2 pm Eastern Time; viewers of the live presentation on May 21 or participants in the live telephone question-and-answer session on June 10 receive 1 CRCE credit hour. To register, call the AARC at (214) 243-2272.

May 24 in Weirs Beach, New Hampshire. The Vermont-New Hampshire Society for Respiratory Care presents its "CE (Continuing Education) Cruise...IV" aboard the Motor Vessel Mount Washington. CRCE credit has been requested. Contact Bill Hay at (603) 267-7406.

May 24 in San Antonio, Texas. The TSRC (Alamo District), in conjunction with the University of Texas Health Science Center at San Antonio, and Wilford Hall Medical Center, announce the 1st Annual Riverwalk Respiratory Symposium at the Marriott Riverwalk. Topics include the future of respiratory care, the impact of managed care, implementing RC protocols, and sleep studies. CRCE credit has been requested. Contact the University of Texas Health Science Center at San Antonio, Department of Respiratory Care, 7703 Floyd Curl Dr, San Antonio TX 78284-7784, (210) 567-3706.

May 1–June 30—Management Training Institute. The deadline to enroll in Module 2 (Communication Skills and Managerial Accounting Part 1) and Module 6 (Business and Health Law and Organizational Behavior) of the Management Training Institute (MTI) Independent Study Program is April 1. MTI is an 8-module program of the AARC and Indiana University. Sign up now to earn the MTI Certificate in Management Development. Call Barbara Hakes at (317) 274-4475.

OTHER MEETINGS

March 28-29 in Rosemont, Illinois. Plutonia Associates Inc, and the Triton College Continuing Education Center for Health Professionals invite you to attend the Neonatal and Pediatric Respiratory Care Conference at the Holiday Inn O'Hare (Holidome). The conference features speakers from the Chicago area addressing mechanical ventilation, trauma, home care, and abuse; a panel discussion on advanced directives is planned. The conference also features exhibits. Continuing education credit has been requested for RCPs, registered nurses, and physicians. Contact Wanda M Manning at (708) 456-0300, extension 3767.

May 10–15 in New Orleans, Louisiana. The American Lung Association/American Thoracic Society hosts its annual International Conference. The conference features information on the prevention, control, and management of lung disease. For more information, write to the 1996 International Conference, ALA/ATS, 1740 Broadway, New York NY 10019-4374.

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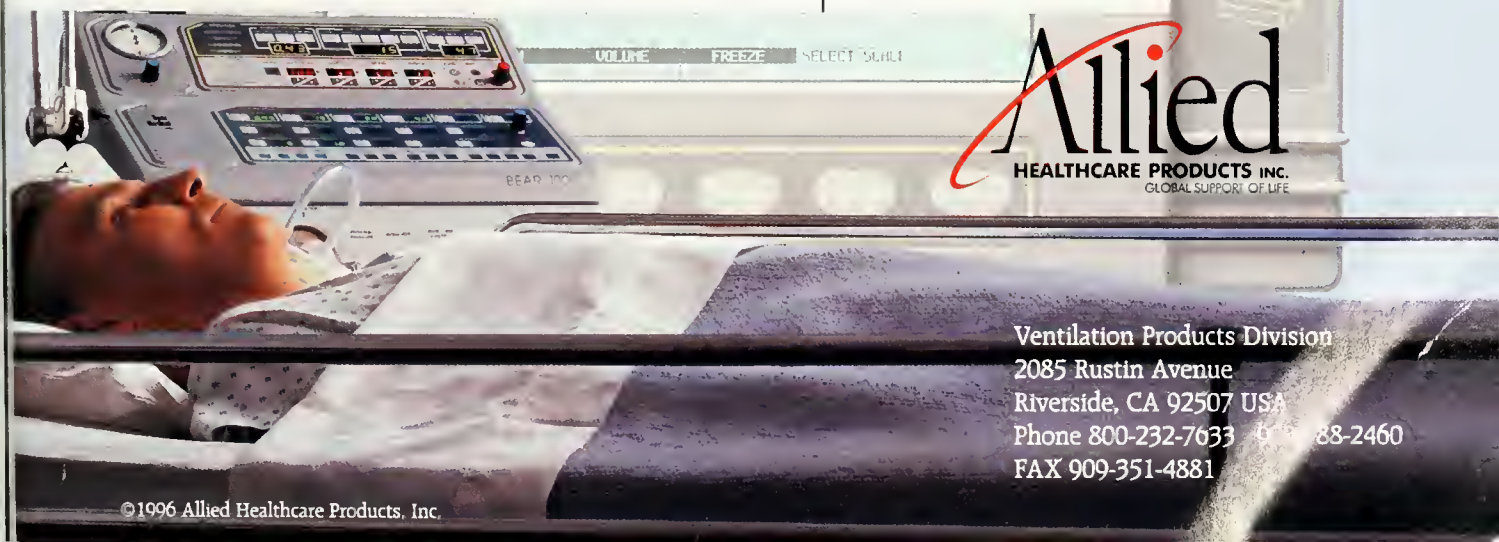

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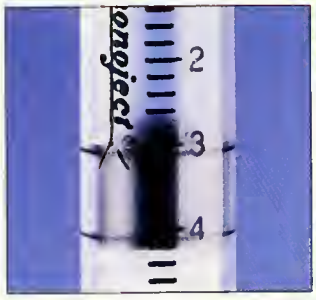

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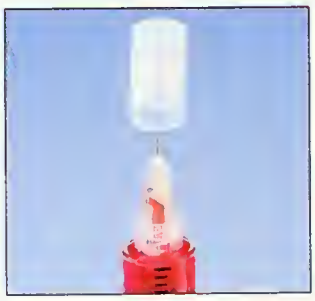

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